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VIOLENCE AMONG OFFENDERS WITH MENTAL DISORDERS

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The cover shows hemlock, a plant associated with both violence and mental disorders. In Ovid's *Metamorphoses*, Juno commissions Tisiphone, one of the Furies, to give the Boeotian king Athamas a potion containing hemlock; after that, he is struck with insanity and kills one of his sons.

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VIOLENCE AMONG OFFENDERS WITH MENTAL DISORDERS

THESIS FOR DOCTORAL DEGREE (Ph.D.)

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20 October 2017, 10.00 am

Livet skræmde meg, og alt var audt.
Alt i verdi tyktest tomt og daudt.
Eg visste *over* meg dei høge salar –
eg var i botnane på djupe dalar
…
Næste dagen var det frå seg sytt.
Livet gledde meg, og alt var nytt.
Eg øygna *under* meg dei høge salar
og elv og botnar i dei djupe dalar.

Olav Aukrust

Åkej. Man är väl fel person. I fel roll. I fel ålder, på fel plats, vid fel tidpunkt.

Hängig på morron. Åkej. Döle vid lunchdags. Färdig vid middan.

Åkej åkej. Det är väl hösten eller vintern eller våren. Nån bristsjukdom.

Jag ska försöka bättra mig. Som den där med nie barn som jobbar heldag och dessutom bakar vörtlimper.

Och verkligen engagera mig. Åkej. Och skriva brev till mormor. Och få en tid hos tandläkarn. Och låna alla böcker som man *måste* läsa ju.

Och börja föra kassabok. Och börja läsa ryska. Och koka äppelmarmelad. Och repetera tyska. Och dammsuga bilen förstås.

Och vara på ett jättefint humör. Åkej åkej. Men det får bli en annan dag. Just nu så har jag inte lust till nånting alls. Nehej. Jag går och knyter mig.

Sonja Åkesson

Like sands through the hourglass,
so are the days of our lives

attributed to Socrates

ABSTRACT

Background. Interpersonal violence is described as a global health problem by the World Health Organization. Even though individuals with severe mental disorders commit only a fraction of all violent acts, they appear to be more likely to behave violently than the general population. Assessments of violence risk may help health care services to better understand how to reduce the likelihood of violent outcomes; consequently, adequate assessment methods are crucial. On the other hand, violent individuals with severe mental disorders constitute a vulnerable group in society, so it is conceivable that they are also subjected to violence themselves. Again, health care services may be made conducive to protection.

Method. The violence risk assessment instruments COVR, LSI-R, HCR-20^{V3}, and SAPROF were applied to a forensic psychiatric sample consisting of 200 detainees undergoing a forensic psychiatric evaluation; after a year, the sample was followed up regarding violence. Rates of self-reported violent victimization and health care utilization were compared between the forensic psychiatric sample and a general population sample consisting of 600 controls matched by age span, sex, and occupation. Finally, rates of violent ideation were examined in the forensic psychiatric sample and a general psychiatric sample consisting of 390 patients at discharge; after 20 weeks, the samples were followed up regarding violence.

Results. In the forensic psychiatric sample, 23.3% committed violent acts in the follow-up year. Correlations between the risk instruments under study were considerable, while the predictive performance was small for COVR (AUC = 0.61), medium for LSI-R (AUC = 0.70), large for HCR-20^{V3} (AUC = 0.79), and large for SAPROF (AUC = 0.78). Violent victimization was reported by 52.3% of the forensic psychiatric sample and 11.1% of the general population sample (*RR* = 8.2), health care utilization by 47.7% and 23.7%, respectively (*RR* = 2.0), and unmet health care needs by 42.2% and 16.7%, respectively (*RR* = 3.4); there was no distinct association between violent victimization and health care utilization. Violent ideation during lifetime was reported by 32.5% of the forensic psychiatric sample and 35.7% of the general psychiatric sample; when both samples were combined, there was an association between violent ideation and ensuing violent acts (*OR* = 2.42), but other performance measures indicated a poorer predictive ability.

Conclusions. Both violent perpetration and violent victimization are common in a forensic psychiatric context. In this context, COVR, LSI-R, and especially HCR-20^{V3} and SAPROF, are methods suitable for predicting violent perpetration, which may in turn facilitate prevention. Health care services should actively improve availability and take measures to protect forensic psychiatric populations from violent victimization. Violent ideation seems to be equally prevalent in forensic psychiatric and general psychiatric populations. When taking a medical history, mental health care professionals should address not only violent ideation, but also other factors that may be associated with violent outcomes. To improve performance of both assessments and interventions, the needs and characteristics of the individual must also be considered.

SVENSK SAMMANFATTNING

Bakgrund. Mellanmänskligt våld drabbar en mängd människor, och beskrivs av Världshälsoorganisationen som ett världsomfattande hälsoproblem. Bara en bråkdel av alla våldshandlingar begås av personer med allvarliga psykiska störningar, men dessa förefaller ändå vara mer våldsbenägna än an andra. Med hjälp av våldsriskbedömningar kan sjukvården bidra till att förebygga våld, och följaktligen är lämpliga bedömningsmetoder av avgörande betydelse. Våldsbenägna personer med allvarliga psykiska störningar utgör emellertid en mycket utsatt grupp i samhället, och det kan antas att de också utsätts för våld. Även i detta avseende kan sjukvården bidra till att våld förebyggs.

Tillvägagångssätt. Instrumenten COVR, LSI-R, HCR-20^{V3} och SAPROF användes för att bedöma risken för våldsutövning i en rättspsykiatrisk urvalsgrupp bestående av 200 häktade personer som var intagna på en utredningsavdelning för att genomgå en rättspsykiatrisk undersökning, efter ett år följdes gruppen upp gällande våldsutövning. Med avseende på förekomsten av utsatthet för våld och sjukvårdskonsumtion jämfördes den rättspsykiatriska gruppen också med en urvalsgrupp från allmänbefolkningen bestående av 600 kontroller som matchats beträffande åldersspann, kön och sysselsättning. Dessutom jämfördes den rättspsykiatriska gruppen med en urvalsgrupp bestående av 390 allmänpsykiatriska patienter som just skrivits ut, denna gång avseende förekomsten av självrapporterade våldstankar; efter 20 veckor följdes grupperna upp avseende våldsutövning.

Resultat. I den rättspsykiatriska gruppen begick 23,3% våldshandlingar under uppföljningsåret. COVR hade en ringa förmåga att förutsäga våld (AUC = 0,61), LSI-R en måttlig (AUC = 0,70), HCR-20^{V3} en god (AUC = 0,79), och SAPROF också en god (AUC = 0,78) förmåga. Utsatthet för våld uppgavs av 52,3% av den rättspsykiatriska gruppen och 11,1% av allmänbefolkningsgruppen (*RR* = 8,2), vårdkonsumtion av 47,7% respektive 23,7% (*RR* = 2,0), och ouppfyllda sjukvårdsbehov av 42,2% respektive 16,7% (*RR* = 3,4). 32,5% av den rättspsykiatriska gruppen och 35,7% av den allmänpsykiatriska gruppen uppgav att de haft tankar på våld någon gång livet; då dessa båda grupper slogs ihop förelåg visserligen ett samband mellan våldstankar och följande våldshandlingar (*OR* = 2,42), men andra mått visade att det näppeligen går att förutsäga våld endast utifrån en uppgift om våldstankar.

Slutsats. I ett rättspsykiatriskt sammanhang är såväl våldsutövning som utsatthet för våld vanligt förekommande. COVR och LSI-R, och framför allt HCR-20^{V3} och SAPROF, kan i detta sammanhang användas för att förutsäga risken för våld, vilket i sin tur ger en möjlighet att förhindra våld. Sjukvården måste bli mer tillgänglig och vidta åtgärder för att skydda psykiskt störda lagöverträdare från att utsättas för våld. Förekomsten av våldstankar förefaller vara ungefär lika stor bland psykiskt störda lagöverträdare och allmänpsykiatriska patienter. Vid anamnestagning bör inte bara våldstankar beaktas, utan även andra omständigheter som kan innebära en förhöjd risk för våldsutövning. Såväl våldsriskbedömningar som våldsförebyggande åtgärder behöver utgå från den enskilde individen för att förbättra utfallet.

LIST OF SCIENTIFIC PAPERS

I. **Persson**, **M**., Belfrage, H., Fredriksson, B., & Kristiansson, M. (2017). Violence during imprisonment, forensic psychiatric care, and probation: Correlations and predictive validity of the risk assessment instruments COVR, LSI-R, HCR-20^{V3}, and SAPROF. *International Journal of Forensic Mental Health, 16*(2), 117-129.

II. **Persson**, **M**., Belfrage, H., & Kristiansson, M. (2017). Violent victimization and health service utilization in a forensic psychiatric context: A comparison between offenders with mental disorders and matched controls. *BMC Psychiatry, 17*(91), 1-10.

III. **Persson**, **M**., Sturup, J., Belfrage, H., & Kristiansson, M. Self-reported violent ideation and its link to interpersonal violence among offenders with mental disorders and general psychiatric patients. *Submitted manuscript*.

CONTENTS

LIST OF ABBREVIATIONS

95% CI	95% confidence interval
AMPA	α-amino-3-hydroxy-5-methyl-4-isoxazolepropionic acid
ANOVA	Analysis of variance
AUC	Area under the ROC curve
COVR	Classification of Violence Risk
DSM	Diagnostic and Statistical Manual of Mental Disorders
FET	Fisher's exact test
GAD	Generalized anxiety disorder
GABA	γ-aminobutyric acid
H_0	Null hypothesis
H_a	Alternative hypothesis
HCR-20^{V3}	Historical, Clinical, Risk Management-20, version 3
HPA	Hypothalamic-pituitary-adrenal
ICD	International Classification of Diseases
ICC	Intraclass correlation
IQ	Intelligence quotient
LR+	Likelihood ratio for positive results
LR-	Likelihood ratio for negative results
LS/CMI	Level of Service/Case Management Inventory
LSD	Lysergic acid diethylamide
LSI-R	Level of Service Inventory, Revised
N or *n*	Number
NMDA	*N*-methyl-D-aspartate
NPV	Negative predictive value
OR	Odds ratio
p	Probability
PANSS	Positive and Negative Syndrome Scale
PCP	Phencyclidine
PD	Personality disorder
PPV	Positive predictive value

PTSD	Post-traumatic stress disorder
Q_1	First quartile
Q_3	Third quartile
ROC	Receiver operating characteristics
RR	Relative risk
SAPROF	Structured Assessment of Protective Factors
SE	Standard error
SILC	Swedish Living Condition Surveys
SMD	Severe mental disorder
SPSS	Statistical Package for the Social Sciences
z	Test statistic for *r* to *z* transformation

1 BACKGROUND

1.1 OFFENDERS WITH MENTAL DISORDERS

1.1.1 A historical survey

From time immemorial, criminal liability has attracted interest. Throughout history, the import of volition has been discussed, sometimes in direct connection with offenders with mental disorders.

This is reflected in the major religions of the world. For example, while the Judeo-Christian *Pentateuch* ordains capital punishment for homicide (Exod. 21:12, 21:23, Lev. 24:17, Deut. 19:21), it also states that a murderer who lacked intent could flee into special cities of refuge instead (Exod. 21:13, Deut. 4:42). The Hindu legal text *Manusmriti* establishes that a king must always forgive ill men who inveigh against him (Manu, 1991), and a passage in the Buddhist *Tripitaka* observes that litigation may be removed on account of insanity (Bodhi & Nanamoli, 2015). In Islam, the outlook is similar; in the hadith collection *Sunan Abu Dawud*, it is stated that the actions of a lunatic will not be recorded until he recovers his wits (Hasan, 2012).

Classical philosophy proffers comparable opinions. In his dialogue *Laws*, Plato wrote that if someone commits a murder while insane, he is to stay in another country for a year (Plato & Pangle, 1988). Aristotle's *Nicomachean Ethics* imparts, perhaps less pithily, that it is useful for legislators to distinguish between voluntary and involuntary actions when assigning punishments (Aristotle, Bartlett, & Collins, 2011). Roman orator Cicero agrees: in the third of his *Stoic Paradoxes*, he argued that crimes are to be measured by the bad intentions of men (Cicero & Edmonds, 1850). Augustine of Hippo, the Church Father, asked how an imbalanced individual with club and cudgel can be called guilty when he does not know what he has done (Robinson, 2013). Later philosophers hold similar views. In *Leviathan*, published in 1651, the Englishman Thomas Hobbes explains that there is no law over mentally deficient people or madmen, because they are not equipped to take it in (Hobbes, 1996). Correspondingly, his compatriot John Locke writes in the 1691 work *Two Treatises of Government* that lunatics and idiots are incapable of knowing the law (Locke, 1988).

Ancient legislation also dealt with disordered offenders. The Babylonian king Hammurabi's law code stele, dating back to the 18th century BC, acquits unwitting perpetrators (Harper, 1904); this section is sometimes construed as a basis for insanity defence (Sperry, 2016). Roman law also stipulated exculpation or mitigation of sentences by reason of insanity, exemplified by Emperor Marcus Aurelius who waived legal proceedings against a lunatic parricide (Monro, 1904). Medieval regulations followed the same pattern. In the somewhat depreciatory terms of that time, the legal treatise *De Legibus et Consuetudinibus Angliae*, completed by English jurist Henry de Bracton in about 1256, holds that a lunatic can no more commit a felony than a brute animal (Diamond & Quen, 1994).

Moreover, some of the pioneers of modern psychiatry took notice of offenders with mental disorders. In the early editions of *Lehrbuch der Psychiatrie*, German psychiatrist Emil Kraepelin defined moral insanity as an innate feeblemindedness impairing the ability to restrain callous satisfaction of egotism; later versions treated the condition as a type of psychopathic personality, not necessarily coinciding with deficient intellectual faculties (Kraepelin, 1904; Wetzell, 2000). Swiss psychiatrist Eugen Bleuler, notable for coining the term schizophrenia, meant that criminal propensity per se was an innate moral defect rather than a disorder, and that this defect occured among both sane and insane persons (Bleuler, 1896; Möller & Hell, 2002). Sigmund Freud, the Austrian neurologist who founded psychoanalysis, wrote in *The Ego and the Id* that many offenders commit crimes to assuage an unconscious sense of guilt by transforming it into something tangible (Freud, Freud, Strachey, Strachey, & Tyson, 1961).

The earliest Swedish records of offenders with mental disorders emanate from the provincial laws, codified in the 13th and 14th centuries. Most of them decreed that the insanity of a subject was to be publicly acknowledged and that the kinsfolk must fetter him; if these steps had been taken beforehand, an assault committed by the subject was regarded as a misadventure for which a fine was imposed (Munktell, 1943), instead of the usual corporal punishment. The *lex Helsingiae* represented a special case, in so far as it allowed the question of insanity to be evaluated *after* the unlawful act (Kjellström, 1909). Before long, the written provincial laws were supplanted by the nationwide country laws passed by Swedish kings Magnus Eriksson in about 1350 and Christopher of Bavaria in 1442. These laws were in turn replaced by the *Civil Code of 1735*, but the provisions regarding impunity for offenders with mental disorders remained essentially the same. In a way, 1826 saw the birth of forensic psychiatry in Sweden, when a new royal statute stipulated that offenders with mental disorders should undergo medical evaluations in order to determine their competency to stand trial (Qvarsell, 1993).

Since the promulgation of the still valid Swedish *Penal Code of 1965*, mental disorders no longer absolve defendants from criminal responsibility (Svennerlind et al., 2010). For that reason, forensic psychiatric evaluations are primarily aimed at sentencing recommendations. In 2016, 470 such evaluations were carried out in Sweden (National Board of Forensic Medicine, 2017). Forensic psychiatric care is a relatively rare sanction: that same year, there were only 293 such sanctions, whereas the number of prison sentences totalled 10,400 (National Council for Crime Prevention, 2017). On the other hand, persons sentenced to care constitute a sizable proportion of psychiatric inpatients; in 2016, 27.4% of 4292 psychiatric hospital beds were occupied by forensic psychiatric patients (Swedish Association of Local Authorities and Regions, 2017), whose median length of stay amounted to approximately 53 months (Nationellt rättspsykiatriskt kvalitetsregister, 2017). The great lengths of stay are due to the fact that forensic psychiatric care not only strives to safeguard the rights of the patients, but also at safeguarding the need for protection in society. Both of these aspects are addressed in this thesis, as it deals with violent perpetration as well as violent victimization in a forensic psychiatric context.

1.2 DEFINITIONS OF VIOLENCE

Although most people are familiar with the meaning of the word violence, it may be appropriate to begin with a definition of the concept. A definition is a description of the meaning of a term. Definitions are divided into partly overlapping sub-types, three of which may be of interest in this context. Typically found in dictionaries, the *lexical definition* merely specifies the function of a word in common usage; for example, weight may be defined as the heaviness of an object. The theoretical or *conceptual definition* provides a more thoroughgoing explanation for scientific use; here weight is the product of the mass of the object and the gravitational acceleration. In order to measure a phenomenon, an *operational definition* must be employed; in this case, weight would be the number displayed on a spring scale below which the object has been hung. These different definitions may also be useful in the field of violence.

1.2.1 Lexical definition

In the *Oxford English Dictionary*, violence is defined as the deliberate exercise of physical force against a person, property. The dictionary also explains that the word is borrowed from French and derives from the Latin *violentia*, use of force, perhaps related to *vis*, force. The etymology of this word is somewhat uncertain, but its origin may be the presumed Proto-Indo-European root **uiH-*, meaning chase or strength (de Vaan, 2008).

1.2.2 Conceptual definition

According to the *Encyclopædia Britannica*, violence is an act of physical force that causes or is intended to cause physical or psychological harm, whereas the wider construct aggression connotes a more general hostile behaviour that may be physical, verbal or passive. The World Health Organization stresses the importance of malice, and defines violence as the intentional use of physical force or power, threatened or actual, against oneself, another person, or against a group or community, that either results in or has a high likelihood of resulting in injury, death, psychological harm, maldevelopment, or deprivation (World Health Organization, 2002).

1.2.3 Operational definition

Operational definitions of violence often resemble the conceptual ones. For example, violence has been defined as acts of battery that resulted in physical injury; sexual assaults; assaultive acts that involved the use of a weapon; or threats made with a weapon (Monahan et al., 2001). However, even though operational definitions aim at facilitating the measurement of violence, they often seem to lack a description of how to obtain data. For example, should information about violence be gathered from assailants, victims, third-party sources, police reports, or crime registries? Unfortunately, this shortcoming hampers comparative evaluations of research findings.

1.3 TYPOLOGY OF VIOLENCE

For decades, several textbooks and articles have categorized aggression as either instrumental or reactive (DiGiuseppe & Tafrate, 2007), and the closely related concept of violence may be divided in the same way. Instrumental violence is perpetrated with a certain goal and may be characterized as premeditated or proactive; whereas reactive violence is impulsive, retaliatory, or induced by emotions (Huss, 2009). Some scientists assert that this typology is out of date (Bushman & Anderson, 2001), and it has been suggested that, rather than being dichotomous, violence has a dimensional structure (Flynn, 2013).

The World Health Organization (2002) recognizes three types of violence, namely self-inflicted violence, interpersonal violence, and collective violence. Self-inflicted violence implies that the perpetrator and the victim are the same individual, as is the case with suicide and some self-injurious behaviour. Interpersonal violence, the main subject of this thesis, refers to violence between individuals. Finally, collective violence is instrumental violence between groups for political, social, or economic reasons; as in warfare, genocide, terrorism, riots, and corporal punishments. The World Health Organization applies an additional classification based on the nature of violent acts; the acts are classified as physical, sexual, psychological, or neglectful. As shown in Figure 1, these types and classes may be illustrated by an organization chart or a table, capturing both the perpetrator-victim relationship and the nature of violence.

Figure 1. Typology of violence.

	Self-Directed		Interpersonal					Collective		
			Family/Partner			Community				
	Suicidal Behaviour	Self-Abuse	Child	Partner	Elder	Acquaint-ance	Stranger	Social	Political	Economic
Physical	✓	✓	✓	✓	✓	✓	✓	✓	✓	✓
Sexual			✓	✓	✓	✓	✓	✓	✓	✓
Psychological			✓	✓	✓	✓	✓	✓	✓	✓
Deprivation or Neglect	✓	✓	✓	✓	✓	✓	✓	✓	✓	✓

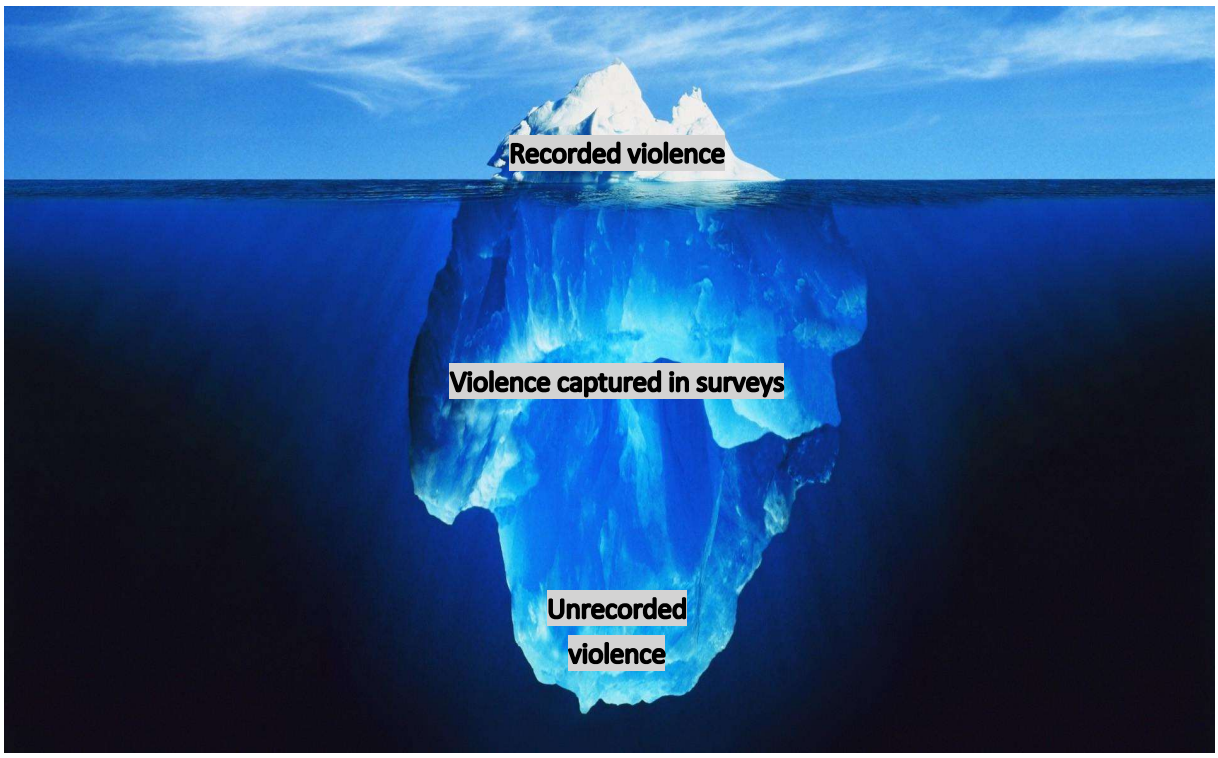
1.4 EPIDEMIOLOGY OF VIOLENCE

Epidemiology refers to the study of the distribution and occurrence of health-related events (Porta, Greenland, & Last, 2008), such as violence. Frequently used epidemiological measures are incidence and prevalence. The incidence if violence is the rate of violent acts in a population during a specific time period, whereas prevalence of violence is the proportion of the population who have experienced violent acts (Renzetti & Edleson, 2008).

Irrespective of measure, gathering epidemiological data is complicated because operational definitions of violence are not concordant, because the number of violent acts may not be commensurate with the number of perpetrators and victims, and because comprehensive sources of data are scarce. Some violence is in most cases recorded by authorities, some is observable only by means of surveys, and some remains unrecorded. For example, the proportion of countries with available police data on the number of homicides ranges from

69% in the Eastern Mediterranean region to 95% in the European region, whereas only 52% of the countries in the world have conducted surveys on sexual violence (World Health Organization, 2014). The iceberg in Figure 2 depicts this predicament, where the large submerged part represents the unrecorded violence. Although hidden to a large extent, violence is without doubt a major contributor to morbidity and mortality worldwide.

Figure 2. Iceberg analogy.



1.4.1 Rates of violence in general populations

As homicides and certain other types of lethal violence are often recorded, they are well suited for international comparisons, and their rates have sometimes been used as proxy levels of overall rates of violent crimes. According to the Global Study on Homicide, the annual homicide rate in the world is between 6 and 7 per 100,000 inhabitants (United Nations Office on Drugs and Crime, 2013), implying that it is not among the top 10 causes of death globally (World Health Organization, 2017). But variations are considerable; almost half of the homicides are committed in countries where just 11% of the global population lives. Homicide rates are above 24 in Southern Africa and Central America, and between 16 and 23 in Middle Africa, South America, and the Caribbean; but only around 2 in Europe and around 1 in Sweden. Variations are substantial also between groups; homicide is the eighth leading cause of death in the black population in the US (Heron, 2016), and the third cause among males aged 15-44 years in the world (World Health Organization, 2014); young males predominate among perpetrators as well. Table 1 provides further details about rates of violence in general populations.

Table 1. Epidemiology of lethal violence in general populations.

	Annual rate (male, female)		Percentage (male, female)		
Age	**Offenders**	**Victims**	**Offenders**	**Victims**	**Population**
Global[a]		6.7 (10.8, 2.5)			
<4		2.7 (2.8, 2.7)			
5-14		1.5 (1.7, 1.2)			
15-29		10.9 (18.2, 3.2)			
30-44		9.3 (15.7, 2.7)			
45-59		6.1 (10.2, 2.0)			
>60		4.5 (6.7, 2.7)			
US[b]	8.7 (15.8, 1.9)	7.8 (12.3, 3.6)	100 (89, 11)	100 (76.5, 23.5)	100 (49, 51)
<14	.2	1.8	.5	4.8	20.5
14-17	14.9	6.4	10.4	5.0	6.1
18-24	29.3	17.0	36.6	23.9	10.8
25-34	15.8	14.2	28.4	28.8	15.7
35-49	7.3	8.6	17.3	22.8	20.5
50-64	3.1	5.1	5.1	9.3	14.2
>65	1.2	3.4	1.7	5.3	12.3
Sweden[ac]		0.9	100 (91, 9)	100 (69, 31)	
<14			0	5	
15-29			46	24	
30-44			34	26	
45-59			15	23	
>60			5	15	
nn				7	

Note. [a]World Health Organization, 2014. Global status report on violence prevention 2014. World Health Organization, Geneva. [b]US Department of Justice, 2011. Homicide trends in the United States, 1980-2008. US Department of Justice, Washington DC. [c]Brottsförebyggande rådet, 2011. Det dödliga våldets utveckling. Fullbordat och försök till dödligt våld i Sverige på 1990- och 00-talet. Brottsförebyggande rådet, Stockholm.

Systematically collected data on non-lethal violence are not available in most countries, but physical and sexual assaults happen every day. The International Statistics on Crime and Justice show that the rate of police-reported assaults is 251 globally and 845.2 in Sweden; and the corresponding rates for rapes are 11.7 and 40.6 (Harrendorf, Heiskanen, & Malby, 2010). These figures do not necessarily reflect the actual facts; countries may differ significantly with respect to both legislation and propensity to report to the police.

1.4.2 Rates of violence in psychiatric populations

Most individuals with severe mental disorders do not commit violent acts (Rueve & Welton, 2008), and those who do make up a rather small proportion of violent offenders (Fazel &

Grann, 2006). Yet, these individuals seem to be more likely to commit violent acts than the general population (K. S. Douglas, Guy, & Hart, 2009; Fazel, Gulati, Linsell, Geddes, & Grann, 2009), even though multiple interacting factors may contribute (Varshney, Mahapatra, Krishnan, Gupta, & Deb, 2016). Violence has been perpetrated by 2-13% of psychiatric outpatients in the past six months to three years (Choe, Teplin, & Abram, 2008), and by 17-20% of inpatients during stays of up to five weeks (Iozzino, Ferrari, Large, Nielssen, & de Girolamo, 2015; Renwick et al., 2016). Rates are higher among forensic psychiatric inpatients, around 30%, but their periods of hospitalization are also longer (L. Bowers et al., 2011; Broderick, Azizian, Kornbluh, & Warburton, 2015). However, violent victimization seems to be more common than violent perpetration among individuals with mental disorders (Choe et al., 2008), with annual rates of 6.4-56.0% (Latalova, Kamaradova, & Prasko, 2014), which is more than eleven times higher than general population rates (Teplin, McClelland, Abram, & Weiner, 2005); lifetime rates are even higher (Cusack, Frueh, & Brady, 2004; Mueser et al., 1998). In correctional settings, the rates of physical victimization for inmates with mental disorders are 1.2-1.6 times higher than for other inmates (Blitz, Wolff, & Shi, 2008); this may indicate that forensic psychiatric populations are particularly hard-stricken.

1.5 AETIOLOGY OF VIOLENCE

The term aetiology refers to the causation of a condition. Accordingly, the principal aim of aetiological research is to assess the cause of the condition, in this case violence. Most scholars agree that the aetiology of violence is multifactorial, and that quite a few aspects must be taken into consideration. Many of these aspects concern violence in general, but are probably germane to psychiatric samples as well.

1.5.1 Biological perspectives

1.5.1.1 Evolutionary aspects

On the Origin of Species (Darwin, 1859) forecasted that the theories of evolution would have applications to future psychology, but it was long before its principles were used in the study of human behaviour (Cartwright, 2016). From an evolutionary point of view, benefits for the species may be promoted not only by cooperation, but also conflict, including violence. For example, violence may increase the ability to defend oneself, to reproduce, to protect offspring, and hence to safeguard the survival of the species (Kurtz & Turpin, 1999). Consequently, violence is believed to have a significant phylogenetic component in both humans and animals. However, although probably underestimated, conspecific lethal violence has been reported to occur in less than 40% of mammalian species; and the rate of conspecific deaths varies greatly even between closely related species—for example 13.27% in lions versus 0.88% in tigers, and 4.49% in chimpanzees versus 0.68% in bonobos. In humans, the rate seems to have varied with time, from 12.08% during the Middle Ages to 1.33% at present (Gómez, Verdú, González-Megías, & Méndez, 2016).

1.5.1.2 Genetic aspects

The clustering of violence within families suggests a substantial genetic impact (Frisell, Lichtenstein, & Långström, 2011). Adoption studies of interpersonal violence show somewhat inconsistent results (Bohman, 1996; Brennan, Mednick, & Jacobsen, 1996; Kendler et al., 2014; Mednick, Gabrielli, & Hutchings, 1984), and some authors point out selective placement factors as possible confounders (Joseph, 2001). However, reviews and meta-analyses establish that genetic influences explain as much as 50% of the variance in aggression (D. R. Miles & Carey, 1997; Tuvblad & Baker, 2011), but also that environmental impact is crucial to the development of a violent phenotype (Laucht, Brandeis, & Zohsel, 2014).

The report of a high prevalence of extra X chromosomes among incarcerated males (Jacobs, Brunton, Melville, Brittain, & McClemont, 1965) sparked an interest in chromosomal and molecular genetic research on violence, though later studies have to some degree dispelled the perception of this karyotype as criminogenic (Carey, 1994). More recent research has focused on the role of monoamine transmitters. For example, violence and aggression may be associated with an increased dopaminergic and a decreased serotonergic activity due to genetic variants coding for the dopamine transporter DAT1 (Guo, Roettger, & Shih, 2007; Vaughn, DeLisi, Beaver, & Wright, 2009), the serotonin transporter 5-HTT (Craig, 2007; Pavlov, Chistiakov, & Chekhonin, 2012; Reif et al., 2007), the dopamine receptor DRD2 (Butovskaya et al., 2013), the serotonin receptor 5-HT2B (Bevilacqua et al., 2010), and the metabolizing enzymes MAOA (Brunner, Nelen, Breakefield, Ropers, & van Oost, 1993; Caspi et al., 2002; Haberstick et al., 2014; Tiihonen et al., 2015) and COMT (Bhakta, Zhang, & Malhotra, 2012; Iofrida, Palumbo, & Pellegrini, 2014). Other genes may also be associated with violence, including CDH13, which encodes the neuronal membrane adhesion protein cadherin 13 (Tiihonen et al., 2015).

1.5.1.3 Prenatal and perinatal aspects

Effects of pregnancy and birth complications appear to be predictive of future criminal behaviour (Hodgins, Kratzer, & McNeil, 2002; Reiss & Roth, 1993), at least when combined with inadequate parenting (Hodgins, Kratzer, & McNeil, 2001). It may be that these complications have an impact on the neuroregulatory systems mediating aggression and violence (LaPrairie, Schechter, Robinson, & Brennan, 2011). Although of borderline significance, this association seems to be present also among individuals with schizophrenia (Cannon et al., 2002).

1.5.1.4 Cell signalling and signalling molecules

As the section on genetic factors implies, *neurotransmitters* may be a contributory cause of violence. These endogenous molecules are typically released from an axon in response to an action potential in the presynaptic neuron. After traversing the synaptic cleft, they bind to receptors on a dendrite, either generating or inhibiting a new action potential in the postsynaptic neuron. *Hormones*—signalling molecules released from endocrine glands and

transported by the circulatory system to specific organs—may also be associated with violence, as well as the loosely defined *cytokines*, which are primarily autocrine, paracrine, or endocrine immunomodulators.

Dopamine is a monoamine derived from phenylalanine or tyrosine. Cells producing dopamine are restricted to a few parts of the brain, but these parts project neuronal pathways reaching many cerebral areas. The nigrostriatal pathway controls motor functions, the mesocortical and mesolimbic pathways modulate motivation and reward, and the tuberoinfundibular pathway regulates the secretion of prolactin from the pituitary gland. Stimulation of dopamine D2 receptors may evoke aggressiveness, which may explain some of the anti-aggressive properties of D2-blocking antipsychotics (Kim, 2004).

Derived from dopamine, the monoamine *norepinephrine* is most importantly synthesized in the locus coeruleus, a brainstem nucleus that is connected with several parts of the central nervous system. Norepinephrine is the primary neurotransmitter of the sympathetic nervous system; in addition, it is secreted from the adrenal glands. The activity of norepinephrine correlates with attention, arousal, and vigilance. An increased activity may elicit aggressive and hostile behaviours in animals and humans (Haden & Scarpa, 2007).

Another monoamine, *serotonin*, is derived from tryptophan. In the central nervous system, serotonin is mainly produced in the raphe nuclei, from which axons reach many parts of the brain. These serotonin pathways regulate several functions, including mood, circadian rhythms, learning, and memory. An inverse relation between serotonin activity and aggression has been hypothesized, but meta-analytical research reveals only a weak correlation of -0.12 (Duke, Bègue, Bell, & Eisenlohr-Moul, 2013); an inverse correlation may be present also among psychiatric patients (Rueve & Welton, 2008).

The monoamines are modulated by other signalling molecules, such as glutamate, GABA, and neuropeptides. *Glutamate* is the principal excitatory neurotransmitter, acting on AMPA and NMDA receptors, as well as other receptors (Meldrum, 2000). It may enhance the excitability of other neural circuits responsible for aggression (Miczek & Fish, 2006). *GABA* is an inhibitory neurotransmitter synthesized from glutamate; around 20% of cortical neurons are GABAergic (Sahara, Yanagawa, O'Leary, & Stevens, 2012). In general, GABA suppresses aggression, but some modulators of the $GABA_A$ receptor may have the opposite effect (Narvaes & de Almeida, 2014). The neuropeptides *oxytocin* and *vasopressin* are produced in the hypothalamus and secreted by the posterior pituitary. Oxytocin may facilitate prosocial behaviour and in that way decrease aggressive behaviour (Lee, 2015), but it may also stimulate maternal aggression (Pedersen, 2013). Vasopressin, which primarily regulates osmolality, also has an inconsistent impact on aggressiveness, at least in animal models (Fodor et al., 2014). Research also suggests that aggression is positively correlated with cerebrospinal fluid *neuropeptide Y* (Coccaro, Lee, Liu, & Mathé, 2012) and *substance P* (Coccaro, Lee, Owens, Kinkead, & Nemeroff, 2012).

Testosterone is a steroid hormone synthesized from cholesterol and secreted from the testicles. Synthesis is regulated through a feedback loop, whereby low testosterone levels stimulate the hypothalamus to release gonadotropin-releasing hormone; the latter hormone stimulates the pituitary gland to release follicle-stimulating and luteinizing hormones, which in turn stimulate testosterone synthesis. By activation of androgen receptors throughout the body, testosterone stimulates growth of muscles, bone, and male reproductive organs. As males are more aggressive than females, testosterone has been presumed to induce aggression, perhaps through its perinatal effect on neural connections (Mazur & Booth, 1998). This association is established in animals (Archer, 1991); significant correlations have also been reported also in humans, albeit with coefficients of only around 0.1 (Archer, Graham-Kevan, & Davies, 2005; Book, Starzyk, & Quinsey, 2001). It may be that testosterone does play a role in promoting aggression, but that it is greatly moderated by a variety of contextual factors (Haller, 2014).

Cortisol, another steroid hormone synthesized from cholesterol, is secreted from the adrenal cortex. Its feedback loop, the HPA axis, resembles that of testosterone; when cortisol levels are low, the hypothalamus produces corticotrophin-releasing hormone, causing the pituitary gland to release adrenocorticotropic hormone, which stimulates cortisol synthesis. Cortisol affects a variety of functions including metabolism and immune response, but its association with aggression is mixed (Huber, Bannasch, & Brennan, 2011), which, however, seems theoretically plausible: as the HPA axis is stress-responsive, cortisol levels are likely to vary depending on whether aggression is proactive or reactive (Tremblay, Hartup, & Archer, 2005).

Other hormones may also influence aggressive behaviour. *Insulin*, secreted by the pancreatic islets, stimulates the cellular uptake of glucose, among several metabolic effects. Finnish research indicates that there may be an association between enhanced insulin levels and violent offending, perhaps due to hypoglycaemia (Ojala, Tiihonen, Repo-Tiihonen, Tikkanen, & Virkkunen, 2015). Thyroid hormones also have metabolic effects, but an increased ratio between *thyroxin* and *triiodothyronine* is linked to aggressive traits and violence (Sinai et al., 2015; Stalenheim, 2004).

Finally, cytokines may be induced by a broad range of aversive factors, such as trauma, microbes, and stress (Korneva & Phelps, 2008). This could explain why aggression seems to be positively correlated with several inflammation-related phenomena, for example levels of *interleukin-10* in plasma (Das et al., 2016), *C-reactive protein* and *interleukin-6* in plasma (Coccaro, Lee, & Coussons-Read, 2014), soluble *interleukin-1* receptor II protein in cerebrospinal fluid (Coccaro, Lee, & Coussons-Read, 2015), and expression of *tumour necrosis factor-alpha* (Suarez, Lewis, & Kuhn, 2002).

1.5.1.5 Brain anatomy and activity

Research employing neuroimaging techniques such as functional magnetic resonance imaging (fMRI), positron emission tomography (PET), and single photon emission computed

tomography (SPECT), suggests that several brain structures are involved in violence and aggression. Lesions as a result of trauma, tumours, metabolic disturbances, and reduction in brain matter may bring about deficits that underlie aggressive behaviour.

In 1848, the unfortunate railway worker Phineas Gage was involved in a detonation accident where a tamping iron penetrated his left frontal lobe, after which his personality was altered and he became fitful, irreverent, and impatient (Guidotti, 2012; Neylan, 1999). His tragic fate may have been the starting point for a enduring interest in the *prefrontal cortex*, a region linked with several cognitive and executive functions (Funahashi, 2017; Funahashi & Andreau, 2013). In the prefrontal and *cingulate* cortices, serotonin appears to modulate and often suppress aggression (Siever, 2008). A decrease in prefrontal activity relative to subcortical activity appears to be associated with impulsive aggression (Bufkin & Luttrell, 2005). Brain imaging studies report that structural and functional deficits located in the right orbitofrontal cortex and left dorsolateral prefrontal cortex are likely to be implicated in violent and antisocial behaviour (Yang & Raine, 2009).

In addition to cortical regions, the limbic system also appears to be involved in aggression and violence, often through the mediation of neuropeptides. Located deep in the temporal lobes, the *amygdala* consists of nuclei receiving input from the sensory systems and sending projections to hypothalamus and other subcortical and cortical structures. The nuclei are thought to contain GABAergic neurons with important inhibitory functions (Nuss, 2015), contributing to an essential ability to perceive and process emotions, including aggression and fear. Several lines of research suggest that the structure is associated with aggressiveness; for example, individuals with reduced amygdala volumes display higher aggression scores (Matthies et al., 2012) and an increased risk of future violence (Pardini, Raine, Erickson, & Loeber, 2014). The *hypothalamus*, located in the diencephalon, provides an important link between the nervous and endocrine systems, with the goal of maintaining homeostasis. Activity in the ventral parts of the hypothalamus seems to be essential to the generation of attack behaviour (Falkner, Grosenick, Davidson, Deisseroth, & Lin, 2016; Falkner & Lin, 2014). Around the cerebral aqueduct, there are neuronal cell bodies forming the *periaqueductal grey*, an interface between the forebrain and nuclei in the brainstem (Benarroch, 2012). It controls pain modulation, but stimulation of certain regions may also result in quiescence as well as defensive responses including aggression (Haller, 2014).

1.5.1.6 Intoxicants

Pervasive evidence corroborates the association between intoxicants and aggression. Both proximal and distal effects of intoxicants may be relevant in this context. However, personality traits, cultural norms, and expected effects among substance users may offer more explanatory power than the mere ingestion.

For example, even though the link between *alcohol* and aggression is epidemiologically established, the underlying neurobiological processes remain poorly understood (Miczek, DeBold, Hwa, Newman, & de Almeida, 2015). Action on the $GABA_A$ receptor seems to be

necessary for alcohol-induced aggression, NMDA and 5-HT3 receptors may also be important action sites (Miczek, Fish, De Almeida, Faccidomo, & Debold, 2004). *Benzodiazepines*, although intended as tranquilizers, may also escalate aggression by acting on certain subtypes of the $GABA_A$ receptor (Newman et al., 2015). Whereas *opiate* intoxication seems to reduce aggression, withdrawal may facilitate it, presumably by activation of dopamine (Miczek et al., 1994). *Amphetamines* are sympathomimetic and may exert a pro-aggressive effect by releasing dopamine (Studer, Näslund, Westman, Carlsson, & Eriksson, 2016), possibly modulated by opioid peptides (Asghar, De Souza, & National Institute on Drug Abuse., 1989); neurotoxic destruction of serotonergic pathways may also contribute (Dawe, Davis, Lapworth, & McKetin, 2009). By blocking reuptake, *cocaine* also contributes to a synaptic accumulation of dopamine (Preedy, 2017), which, along with other complex processes, may elicit aggression (Morton, 1999). In a sometimes tempestuous debate, *cannabis* has been attributed both pacifying and inciting properties; this suggests a biphasic effect, whereby smaller doses stimulate aggressive behaviour and larger quantities are inhibitory (Mechoulam, 2002). Undeniably, studies do report that cannabis use is a significant predictor of violence in the general population (Norström & Rossow, 2014), among persons with severe mental illness (Dharmawardene & Menkes, 2017), and among delinquents (Schoeler et al., 2016). However, explanatory mechanisms remain somewhat elusive, but it may be that cannabinoid receptors on GABA neurons in the amygdala play a crucial role (ElSohly, 2007). Research on the association between psychedelics and aggression is inconclusive, but the NMDA receptor agonist *PCP* may pronounce aggressive tendencies (Audet, Goulet, & Doré, 2009); also, *LSD* may intensify defensive reactions, an effect perhaps modulated by serotonergic neurons in the raphe nuclei (Miczek, 1987). Furthermore, *anabolic steroids* appear to promote violent outcomes, (Beaver, Vaughn, Delisi, & Wright, 2008; Klötz, Petersson, Isacson, & Thiblin, 2007; Thiblin & Pärlklo, 2002), perhaps by altering levels of serotonin and vasopressin, and modulating of $GABA_A$ receptors (Oberlander & Henderson, 2012).

1.5.2 Psychological perspectives

Numerous psychological theories have been posited to describe and offer explanations for violent and aggressive behaviour.

Innate urges and dispositions are emphasized in the *instinct theories*. Austrian physician Alfred Adler introduced the concept of an aggression instinct in 1908; the concept was originally met with disapproval from his colleague and fellow countryman Sigmund Freud, who regarded aggression as a reaction to thwarted libidinal force (Corsini & Wedding, 2010). Fifteen years later, Freud did however tacitly endorse Adler's theory by suggesting a death force, whose aggressive energy was redirected away from the self (Baron & Richardson, 1994). In the 1960s, Nobel Prize winner Konrad Lorenz also embraced instinct theory, but from an ethological rather than psychoanalytical point of view (Greenberg & Haraway, 1998).

Soon, the intrapsychological approach of the instinct model was criticized, and new theories asserted that aggression stems from elicited *drives*, that is non-instinctual motivational forces induced by deprivation. In 1939, a group of Yale scholars propounded the *frustration-aggression hypothesis* (Dollard et al., 1939). It states that aggression always presupposes frustration and that frustration always generates aggression; frustration develops when goal-directed activities are blocked. Although meritoriously straightforward, the hypothesis did not seem to be borne out empirically (Goldstein, 1981). Its core tenet was, however, retained when the subsuming *cognitive neoassociation theory* was formulated. According to this theory, frustration generates aggression only if it produces negative affect (Berkowitz, 1989). This affect activates ideas, memories, and physical discomfort, which are associated with tendencies to attack or escape and which can be suppressed or intensified by subsequent cognitive processes (Berkowitz, 1990). Dolf Zillman's *excitation-transfer theory* may be regarded as another take on the drive approach; it states that excitation aroused by a stimulus may be cognitively transferred—or rather misattributed—to other stimuli (Zillman, 1988).

Canadian-American psychologist Albert Bandura also agreed that aggression could not be explained solely by either instincts or drives. In his influential bobo doll experiments, he observed that children imitated an adult model who clubbed a toy with a mallet and then was rewarded (Bandura, Ross, & Ross, 1961, 1963). His *social learning theory* proposes that aggressive behaviour is acquired by observational learning and mediated by cognitive processes (Bandura, 1973, 1977). When tested empirically, considerable variation in effect sizes has been shown (Pratt et al., 2010). Another theory emphasizing learning processes is the *information processing model*: aggressive scripts are acquired through observational and enactive learning, and activated through memory and cues (Huesmann, 1988). Aiming at providing an integrative framework, the *general aggression model* suggests that personal and situational input acts through an internal state comprising cognition, affect, and arousal, and influences appraisal and decision, and, finally, actions (C. A. Anderson & Bushman, 2002).

1.5.3 Social and criminological perspectives

The Enlightenment of the 18th century paved the way for the *classical school* of criminology, founded by utilitarian philosophers Cesare Beccaria and Jeremy Bentham. Contrary to previous beliefs about divine predestination, the movement points out the paramount importance of free will regarding delinquency. The classical school fell from favour in the late 1800s, when it was challenged by new theories (Vito & Maahs, 2017). However, its traditions were continued in the subsidiary *neoclassical school*, which more pronouncedly takes contextual circumstances into account (Lilly, Cullen, & Ball, 2015). For example, the *rational choice theory* assumes that crimes are deliberate and motivated by either gain of benefits or avoidance of adversities (Cornish & Clarke, 2014); the reasoning may be applicable to violent crimes, too (Matsueda, Kreager, & Huizinga, 2006). The *routine activities theory* (L. E. Cohen & Felson, 1979) can be thought of as a more elaborate version of the proverb that the opportunity makes the thief; it assumes not only a motivated offender, but also the presence of a target and the absence of guardians.

The 19th century saw the rise of another criminological approach, the *positive school*. French philosopher Auguste Comte, a founding father of positivism and sociology, claimed that science must rest on empirical ("positive") observations and aim at discovering the laws that govern nature and society (Aldridge, 2013). This is summarized in his precept *savoir pour prévoir, prévoir pour pouvoir* (Ray, 1999); knowledge serves prediction, prediction serves power. Another Frenchman, Émile Durkheim, expanded on the ideas; in 1897, he coined the concept of *anomie*, denoting a collapse of social solidarity or derangement of the uniting bonds of collective society (Lilly et al., 2015). This phenomenon may be a breeding ground for the emergence of criminal and violent activity (Bohm & Vogel, 2015), although some scholars contend that Durkheim himself disavowed this connection (Grant, 2002). Columbia University sociologist Robert Merton undoubtedly linked anomie with crime and deviance; his *strain theory* implies that social meritocratic structures exert anomic pressure on some individuals to engage in nonconformist conduct, including crime (Maguire, Morgan, & Reiner, 2012; Merton, 1938); later adaptions of the theory also incorporate violent crimes (Agnew & Kaufman, 2010). While explanatory in nature, the theory has some empirical support (Eitle, 2010).

The *Chicago school* of ecology advanced the idea that social disorganization engenders criminality. It regarded the tremendous growth of the city—rivetingly depicted in Upton Sinclair's 1906 novel *The Jungle*—as a burdensome yet natural ("ecological") event, and found that its accompanying transitory relationships coincided with high crime rates (Tierney, 2009). A source of inspiration for the school was *symbolic interactionism*, conceived by sociologists George Herbert Mead and Charles Cooley at the time of the last turn of the century. The theory rests on the premise that individuals act towards objects on the basis of the meaning they attach to them; this meaning arises out of social interaction and is modified through interpretive processing (Blumer, 1969). The result is a self-fulfilling prophecy, implying that individuals will behave in a manner consistent with the way in which they believe they are viewed by others (Miller, 2009). Thus, the theory may explain how delinquents develop their criminal identities (Eglin & Hester, 2017). Drawing on the same reasoning, the *labelling theory* states that individuals finally act out the role of criminals if they are constantly regarded as criminals by the surrounding society. The theory has been criticized for insufficient empirical corroboration (Knutsson, 1977), but later research has improved its scientific foundation (Farrington & Murray, 2014). Sometimes loosely placed in the Chicago school tradition is Edwin Sutherland's *differential association theory* (Morrison, 1995). Among its nine key principles are that an individual learns criminal attitudes in interaction with others within intimate groups; if attitudes favouring violation of the law dominate, the individual will become delinquent (Sutherland, 1939).

1.5.4 Association between mental disorders and violence

Meta-analyses report that 18.5% of individuals with *psychosis* are violent (Witt, van Dorn, & Fazel, 2013), corresponding to a 49-68% increased likelihood, *OR* = 8.9, 95% CI [5.4, 14.7] (K. S. Douglas et al., 2009); but the association is considerably weaker if there is no

substance abuse comorbidity, *OR* = 2.1, 95% CI [1.7, 2.1] (Fazel et al., 2009). Psychoses may also be associated with repeat offending (Fazel & Yu, 2011). A substantial proportion of patients with first-episode psychosis, 34.5%, commit violent acts before treatment (Large & Nielssen, 2011), and among inpatients, schizophrenia is associated with aggression (Dack, Ross, Papadopoulos, Stewart, & Bowers, 2013).

There is some meta-analytic evidence that individuals with *bipolar disorders* commit more violent crimes than general population controls, *OR* = 4.6, 95% CI [3.9, 5.4], but risk increase is reduced for patients without comorbid substance abuse, *OR* = 2.0, 95% CI [1.4, 2.6] (Fazel, Lichtenstein, Frisell, et al., 2010; Fazel, Lichtenstein, Grann, Goodwin, & Långström, 2010). Three-fold increased odds of violent crimes have been reported for individuals with *depression*; with rates significantly elevated also for those without substance abuse (Fazel et al., 2015). However, some scholars argue that treatment effects must be taken into account (Furukawa, 2015), such as use of antidepressants (Menkes & Herxheimer, 2015). Violent and *suicidal* behaviours are also intertwined (Haglund et al., 2016; Moberg et al., 2014; Stenbacka, Moberg, Romelsjö, & Jokinen, 2012). For example, suicide attempts and threats are significantly and independently associated with violence among both females and males with schizophrenia (Witt, Hawton, & Fazel, 2014).

Another meta-analysis reports an association between *personality disorders* and violent outcomes, *OR* = 3.0, 95% CI [2.6, 3.5], a substantial risk increase (Yu, Geddes, & Fazel, 2012). The association is especially salient as for antisocial and borderline disorders, both belonging to cluster B (Fountoulakis, Leucht, & Kaprinis, 2008). Not included in either DSM or ICD, psychopathy constitutes a special case. This clinical construct is traditionally characterized by arrogance, callousness, irresponsibility, impulsive behaviour, and lack of conscience; features associated with a tendency to violate social conventions and a high risk for violence and aggression (Hare, 1999, 2006).

Prevalence studies do not conclusively support the notion that the risk of violence is increased among persons with *autism spectrum disorder*s (Im, 2016), but in the individual case, the disorder may influence a person to commit violent acts (Allely et al., 2017). *ADHD* is frequent in offender populations, and a history of ADHD symptoms may predict violence (Young, Moss, Sedgwick, Fridman, & Hodgkins, 2015), perhaps reactive rather than proactive violence (Retz & Rösler, 2009). However, some research suggests that it is a history of ADHD *and* aggression that predicts future violence rather than ADHD alone (Andrade, 2009). Aggression is a recognized behavioural manifestation in advancing *dementia*, and it has been estimated that rates of violence against caregivers exceed 20% (Wharton & Ford, 2014). Pooled odds estimates indicate an increased risk of partner violence perpetration among persons with *GAD* and *PTSD*, but the risk of victimization is even more pronounced (Oram, Trevillion, Khalifeh, Feder, & Howard, 2014). Rates of violence and aggression among individuals with *intellectual disability* vary substantially across studies, ranging from 2% to 51% (Bohnen, Bakala, Thakker, & Wijeratne, 2013; Emerson et al., 2001). Individuals with *brain injuries* seem more likely than general population controls to

commit violent crimes, *OR* = 3.3, 95% CI [3.1, 3.5] (Fazel, Lichtenstein, Grann, & Långström, 2011).

1.5.5 Risk factors

Most studies on human violence are observational rather than experimental. This implies that the association between the putative cause and violence might instead be due to random errors, systematic errors, or confounders. Consequently, a broader range of variables associated with violence must be taken into account. These variables—risk factors—can be either causal factors or mere markers.

Risk factors may be defined as the measurable characteristics that a person in a specified population has, that precede the outcome of interest, and that can be used to divide the population into groups (Kraemer et al., 1997). There are several ways to categorize risk factors, the division into causes and markers has already been mentioned. More commonly, the factors are classified by their ability to vary over time. Static factors are fixed and do not change either spontaneously or after treatment. On the other hand, dynamic factors, such as current psychiatric symptoms and signs, are changeable and often amendable to interventions. Thus, they are predictive, yet not necessarily explanatory, constructs (Ward, 2016).

One of the most replicated static risk factors is previous *violent perpetration*, also among individuals with mental disorders; some research indicates that the risk increases linearly with the number of past perpetrations (Rueve & Welton, 2008). *Young adult age* and *male sex* are conceivable risk factors as offending rates seem to peak among men in middle and late adolescence (Maclean & Beak, 2012), but evidence is inconsistent in psychiatric samples (National Collaborating Centre for Mental Health, 2015). *Childhood abuse* and neglect may portend future aggressive or violent behaviour (Al Odhayani, Watson, & Watson, 2013; Lansford et al., 2007; Minh et al., 2013; Pollock et al., 1990; Spatz Widom, 1989); social learning theories and attachment theories provide possible explanations for this association (Dodge, Bates, & Pettit, 1990). As mentioned, static factors cannot change, but the risk they produce may be mitigated by interventions (K. S. Douglas & Kropp, 2002).

Impulsiveness has often been considered a prominent dynamic risk factor (K. S. Douglas & Skeem, 2005). Broadly, it may be defined as a lack of behavioural control and care for the future (Howard, 2017). Even though impulsiveness may seem conceptually related to certain types of violence, there is limited empirical support for this association among individuals with psychosis (Bjørkly, 2013). The *magnitude of psychiatric symptoms*—especially psychotic symptoms—is another dynamic factor. Violent behaviour does not have to be directly motivated by psychotic symptoms (Peterson, Skeem, Kennealy, Bray, & Zvonkovic, 2014), but a meta-regression study has demonstrated a correlation between PANSS scores and violence (Witt et al., 2013). Some studies suggest that the extent of not only positive, but also negative symptoms, may increase risk (Mirzakhanian et al., 2017; O'Reilly et al., 2015). Moreover, *violent ideation* may be related to violent and aggressive outcomes both in the

general population (Nagtegaal, Rassin, & Muris, 2006; Watt, Kohphet, Oberin, & Keating, 2013), among general psychiatric patients (Grisso, Davis, Vesselinov, Appelbaum, & Monahan, 2000; Murray, Eisner, Obsuth, & Ribeaud, 2017), and offenders with mental disorders (Bjerrum Moeller, Gondan, & Novaco, 2017; Daff, Gilbert, & Daffern, 2015). Negative and *antisocial attitudes* may also be pertinent to violence. Meta-analytical research indicates that such attitudes constitute an important risk factor among offenders with mental disorders (Bonta, Blais, & Wilson, 2014). Lastly, *treatment adherence and response* are among the dynamic factors for individuals with mental disorders (Elbogen, Van Dorn, Swanson, Swartz, & Monahan, 2006). Some scholars argue that the concept of dynamic risk factors is encumbered by a conflation of predictive and explanatory entities, and that causal mechanisms must be elucidated to improve usability (Klepfisz, Daffern, & Day, 2016; Ward & Beech, 2015).

1.5.6 Protective factors

Researchers disagree about whether protective factors constitute a distinct concept in its own right, or just connote the absence of risk factors (Office of the Surgeon General, 2001). Different mechanisms by which protective factors operate have been suggested. Whilst direct protective factors predict a low probability of future violent perpetration without taking other factors into account, buffering protective factors attenuate the impact of risk factors (de Vries Robbé, 2014; Lösel & Farrington, 2012). Protective factors may also be divided into internal factors such as *intelligence* (Ttofi et al., 2016), motivational factors such as *motivation for treatment* (Howells et al., 2005), and external factors such as *stable relationships* and *employment* (Walker, Bowen, & Brown, 2013).

1.5.7 Amalgamating dimensions

The foregoing rhapsodic exposition of aetiological perspectives, risk factors, and protective factors does not aim at being entirely comprehensive, but rather at demonstrating that the causes of violence are complex and to a great extent constitute a terra incognita. This implies that several aspects must be taken into account to assess violence, and that this assessment is subject to some uncertainty. For example, in order to predict, and ultimately prevent, violence, several factors must be identified, considered, selected, combined, and translated into a risk estimate. This process is referred to as a violence risk assessment.

1.6 VIOLENCE RISK ASSESSMENTS

The term risk refers to a probability of an outcome within a population, and risk assessment to the use of risk factors to evaluate that risk on an individual level. To assess risk, it is important to define outcomes, measures, samples, risk factors, and analytic procedures (Kraemer et al., 1997; Monahan & Skeem, 2014).

The first generation of violence risk assessment, the *unstructured clinical evaluation*, is based solely on the judgment of the professional. Indisputably, the method is flexible and inexpensive, but also theoretically unsound, as it is susceptible to bias, subjectivity, and

inconsistency. Some research has reported that the accuracy of the method is better than chance (Lidz, Mulvey, & Gardner, 1993), but based on its inferiority to other methods (Ægisdóttir et al., 2006) and its theoretical disadvantages, current literature discourages its use (Scurich, 2016).

Actuarial models gave rise to risk assessments of the second generation. The somewhat abstruse term is derived from the way in which insurance companies use a statistical algorithm to translate the occurrence of static risk factors into an exact probability of a certain outcome (Cautin & Lilienfeld, 2015). Risk factors are precontrived, and the use of an instrument is mandatory; the assessment is exclusively based on the score it produces. The method is empirically well-founded and quite user-friendly, but its static design does not allow for adjustment over time and makes it difficult to identify reversible risk factors. For that reason, it is sometimes described as focusing on prediction rather than prevention (Whittington et al., 2013).

Advantages of the previous methods were combined and revised to develop a third generation of violence risk assessments, the *structured professional judgment approach*. As the name suggests, it is based on the judgment of the professional, but with the guidance of an instrument. Instruments contain empirically supported risk factors, both static and dynamic. At first the assessments were essentially premised on group-based estimates, but in recent instruments—sometimes referred to as fourth generation instruments—factors at the individual level are of substantial importance; this facilitates the application of interventions aligned with the risk in the separate case. Thus, the approach may be described as having a focus on prevention (Whittington et al., 2013).

Differences between actuarial models and the structured professional judgment approach may be thought of as the opposite poles of a continuum rather than a dichotomous structure. The different approaches and aspects may be exemplified by COVR, LSI-R, HCR-20^{V3}, and SAPROF; four of the most utilized assessment instruments in the world (Singh et al., 2014).

1.6.1 COVR, Classification of Violence Risk

The software COVR (Monahan et al., 2005) is an actuarial instrument designed to assess the violence risk among general psychiatric patients; it was launched in 2005 after an 18 year development period (Monahan et al., 2006). This actuarial method is capable of assessing the 40 most predictive risk factors that emerged in the MacArthur violence risk assessment study (Otto & Douglas, 2010), these factors are shown in Table 2. However, due to an iterative classification tree model (Monahan et al., 2000), the software considers only those factors that are relevant in the individual case; the rater reads a question verbatim from the computer screen, and the answer of the patient determines the next question. The rater should confront the patient when an answer disagrees with other information, but if the discrepancy is not resolved, the item should be omitted. At the end of the interview, the software classifies the risk as *very low*, *low*, *medium*, *high*, or *very high*; corresponding to expected violence rates of 1%, 8%, 26%, 56%, and 76%, respectively. Previous research has demonstrated a rather wide

range of AUCs, from 0.58 to 0.77, in general psychiatric and forensic psychiatric samples (Doyle, Shaw, Carter, & Dolan, 2010; McDermott, Dualan, & Scott, 2011; Singh, Serper, Reinharth, & Fazel, 2011; Snowden, Gray, Taylor, & Fitzgerald, 2009; Sturup, Monahan, & Kristiansson, 2013).

Table 2. Risk factors of COVR.

The 40 predictive risk factors included in COVR		
Legal status	Drug abuse	Suicide threat
Psychiatric diagnosis	Gender	Paternal arrest
Prior arrests	Child abuse	Extent of imagined violence
Child abuse	Antisocial PD	Age at first hospitalization
Psychosis	Coercion	Present depression
Loss of consciousness	Prior violence	Years of education
Age	Impulsivity	Hallucinations
Anger	Parental fights	Functioning
Prior arrests	Homicide attempt	Primary diagnosis
Employment	Sexual abuse	Present decompensation
Imagined violence	Marital status	Present substance abuse
Paternal drug use	Threat/control	Present personal problems
Paternal presence	Prior hospitalization	
Alcohol abuse	Imagined self-harm	

1.6.2 Level of Service inventories

Based on the social learning theory of criminal behaviour, the Level of Service inventories have been developed by Canadian psychologists Don Andrews and James Bonta during the past four decades. The inventories are organized around the so called "Central Eight" risk factors, made up by the important "Big Four"—criminal history, antisocial attitudes, antisocial peers, and antisocial personality—together with work, family, leisure, and substance abuse. An outline of Level of Service items is provided in Table 3.

Originally named the Level of Supervision Inventory, the instrument was developed in the late 1970s and early 1980s (Arnold, 2007; Barton-Bellessa, 2012) in collaboration with the Ottawa probation and parole offices (Stevenson & Wormith, 1987). The first pilot form was a 25-page interview schedule, whereas the 58-item sixth version was finally introduced and implemented in 1982 (Andrews, Bonta, & Wormith, 2010). Initial research showed considerable correlations between different raters, $r = 0.80\text{-}0.94$, and between LSI scores and criminal recidivism during probation, $r = 0.47$ (Andrews, 1982).

With minor revisions, the sixth LSI version became the LSI-R, Level of Service Inventory-Revised (Andrews & Bonta, 1995). According to the user's manual, it is composed of 54 static and dynamic items, answered with either a *0* to *3* rating or with *yes* or *no*. As displayed in Table 3, the items are grouped into ten subcomponents. Administration of the instrument

should be based on offender interviews and file information; collateral interviews may also be considered. After completion, a total score is produced. This score constitutes the most important part of the assessment, but the subcomponents and single items may also be relevant. The LSI-R may be used for monitoring offender risk during supervision or treatment, for making probation decisions, and for assessing the likelihood of criminal recidivism. Meta-analytic research has reported that the LSI-R correlates with general recidivism, $r = 0.37$, and violent recidivism, $r = 0.26$ (Gendreau, Goggin, & Smith, 2002).

Table 3. Risk factors of LS instruments.

	LSI-VI	**LSI-R**	**LS/CMI**
Section	Number of items	Number of items	Number of items
1. General factors	**58**	**54**	**43**
Criminal History	10	10	8
Education/Employment	10	10	9
Financial	2	2	-
Family/Marital	4	4	4
Accommodation	3	3	-
Leisure/Recreation	2	2	2
Companions	5	5	4
Alcohol/Drug problems	9	9	8
Emotional/Personal	5	5	-
Probation Conditions	4	-	-
Attitudes/Orientation	4	4	4
Antisocial Pattern	-	-	4
2. Special factors	-	-	**35**
Personal	-	-	14
Criminal History	-	-	21
3. Institutional factors	-	-	**12**
Incarcerations	-	-	9
Complications	-	-	3
4. Other client issues	-	-	**21**
5. Responsivity	-	-	**11**

Two years after its inception, the LSI gave rise to the Youth Level of Service Inventory (Andrews, Robinson, & Hoge, 1984); later on, the Young Offender-Level of Service Inventory (Shields, 1990) followed. The same year that LSI-R was published, an eight-item screening version, LSI-R:SV, was also released (Andrews & Bonta, 1995). The screening version score has been shown to be modestly correlated with recidivism among offenders in general, and some research indicates an ability to predict offences among offenders with mental disorders, AUC = 0.67, $p < 0.001$ (Ferguson, Ogloff, & Thomson, 2009).

After the LSI-R, the LS scales have been revised further. Input from the users of the instrument demonstrated a need to supplement the risk-need assessment with a case management formulation, which led to the preliminary Ontario version, LSI-OR (Andrews, Bonta, & Wormith, 1995), and ultimately to the similar Level of Service/Case Management Inventory, LS/CMI, currently in use (Andrews, Bonta, & Wormith, 2004). LS/CMI comprises eleven sections, but only the first section, containing 43 general LSI-R items covering the central eight factors, contributes to the total score. The specific items of the next four sections cover personal problems and criminal background, prison experience and institutional factors, social and health problems, and responsivity considerations. In the following five sections, the rater carries out risk/need summaries and profiles, decides upon institution or program, and prepares a case management plan. The final two sections, progress record and discharge summary, are not included in the initial assessment but are completed later on. Research indicates that LS/CMI scores are positively correlated with general recidivism, $r = 0.41$, and violent recidivism, $r = 0.29$ (Andrews, Bonta, & Wormith, 2006).

1.6.3 HCR-20, Historical Clinical Risk Management-20

The first version of HCR-20 (Webster, Eaves, Douglas, & Wintrup, 1995) pointed out the relevance of considering score summaries in order to improve prediction accuracy; in other words, the instrument had certain actuarial features. Each item was to be scored *0*, *1*, or *2*, generating a total overall score. However, as it also stressed the importance of taking clinical opinions into account, it may nevertheless be regarded as one of the earliest structured professional judgment instruments (K. S. Douglas, 2014). Preliminary retrospective research showed a correlation of 0.44 between items H2 through C5 combined and violence (K. S. Douglas, Webster, & Wintrup, 1996; Wintrup, Coles, Webster, & Hart, 1994), and of 0.20 between the total HCR-20 score and violence (Wintrup, 1996).

A need of clarifying administration and coding procedures led to the second version of HCR-20 (Webster, Douglas, Eaves, & Hart, 1997). Items were to be coded on a scale of *0*, *1*, and *2*. The recommended way to make the final risk judgment was to use a *low*, *moderate*, and *high* structure; but in addition it was possible to note the total score on the coding sheet. Some items from the previous version were changed, as demonstrated in Table 4. In a review with meta-regression analyses, the instrument produced a median AUC of 0.70 (Singh, Grann, & Fazel, 2011).

Currently, the third version of HCR-20 (K. S. Douglas, Hart, Webster, & Belfrage, 2013) is in use. The rationale for the revision was to reflect feedback from users and contemporary scholarship, and to pay more attention to the decision-making process (K. S. Douglas et al., 2014). Unlike previous versions, version 3 uses a *no*, *partially*, and *yes* rating structure; and the final risk judgment is exclusively communicated as *low*, *moderate*, or *high*. Again, some items were changed, but more importantly, steps were added in the administration process. After (1) assembling information and (2) assessing whether risk factors are present, the evaluator has to (3) determine the relevance of factors, (4) integrate the information into an explanatory framework for the individual being evaluated, (5) specify possible future

scenarios, (6) formulate management strategies giving heed to monitoring, supervision, treatment, and victim safety planning, and (7) to summarize concerns in a conclusory opinion. AUCs typically range from 0.68 to 0.86 in general psychiatric, forensic psychiatric, and offender samples (Hogan & Olver, 2016; Strub, Douglas, & Nicholls, 2014).

Table 4. Risk factors of the three versions of HCR-20.

	HCR-20^{V1}	**HCR-20^{V2}**	**HCR-20^{V3}**
	Historical scale	**Historical scale**	**Historical scale**
H1	Previous violence	Previous violence	Violence
H2	Age at first violent offence	Young age at first violent incident	Other antisocial behaviour
H3	Relationship stability	Relationship instability	Relationships
H4	Employment stability	Employment problems	Employment
H5	Alcohol or drug abuse	Substance use problems	Substance use
H6	Mental disorder	Major mental illness	Major mental disorder
H7	Psychopathy	Psychopathy	Personality disorder
H8	Early maladjustment	Early maladjustment	Traumatic experiences
H9	Personality disorder	Personality disorders	Violent attitudes
H10	Prior release or detention failure	Prior supervision failure	Treatment or supervision response
	Clinical scale	**Clinical scale**	**Clinical scale**
C1	Insight	Lack of insight	Insight
C2	Attitude	Negative attitudes	Violent ideation or intent
C3	Symptoms	Active symptoms of major mental illness	Symptoms of major mental disorder
C4	Stability	Impulsivity	Instability
C5	Treatability	Unresponsive to treatment	Treatment or supervision response
	Risk management scale	**Risk management scale**	**Risk management scale**
R1	Plan feasibility	Plans lack feasibility	Professional services and plans
R2	Access	Exposure to destabilizers	Living situation
R3	Support and supervision	Lack of personal support	Personal support
R4	Compliance	Noncompliance with remediation attempts	Treatment of supervision response
R5	Stress	Stress	Stress or coping

Several other risk assessment instruments have been modelled on HCR-20, such as SARA, Spousal Assault Risk Assessment Guide (Kropp, 1995), and SVR-20, Sexual Violence Risk-20 (Boer, Hart, Kropp, & Webster, 1997)

1.6.4 SAPROF, Structured Assessment of Protective Factors for Violence Risk

Like HCR-20, the SAPROF is based on the structured professional judgment approach, but as the name implies, it is directed towards protective factors rather than risk factors. Its authors saw a need for a guideline to assess these factors, and initially, the Dutch language research version SAPROF-RV (de Vogel, de Ruyter, & Bouman, 2004) was issued in the Netherlands. The selection of its 16 items was based on clinical experience and on literature study of protective and contextual factors. A retrospective study demonstrated a high interrater reliability, ICC = 0.90, and substantial predictive capacity, AUC = 0.19 (de Vries Robbé, de Spa, & de Vogel, 2007).

Table 5. Protective factors of previous research version of SAPROF and the current version.

SAPROF-RV	SAPROF
Historical items	**Internal items**
1. Intelligence	1. Intelligence
2. Attachment to >1 prosocial adult in childhood	2. Secure attachment in childhood
Clinical items	3. Empathy
3. Empathic skills	4. Coping
4. Positive attitude to intervention/authority	5. Self-control
5. Medication/medication adherence	**Motivational items**
6. Resilience/coping skills	6. Work
7. Religion/philosophy of life	7. Leisure activities
Risk management items	8. Financial management
8. Daily structure/work	9. Motivation for treatment
9. Leisure activities	10. Attitudes towards authorities
10. Prosocial supportive network	11. Life goals
11. Stable intimate relationship	12. Medication
12. Mental health professional in network	**External items**
13. Intensive supervision/external control	13. Social network
14. Housing	14. Intimate relationship
15. Finances	15. Professional care
16. Satisfying sexual relationship with adult	16. Living circumstances
	17. External control

As a result of user feedback and updated literature search, the instrument was revised to allow for clinical use. After an initial Dutch language version (de Vogel, de Ruiter, Bouman, & de Vries Robbé, 2007), the current English language version (de Vogel, de Ruiter, Bouman, & de Vries Robbé, 2009) was launched. Differences between the versions are demonstrated in Table 5. In the current version, the 17 items are to be coded on a *0*, *1*, and *2* scale. The final protection judgment is rated as *low*, *moderate*, or *high*, but a more recent edition proposes *low*, *low-moderate*, *moderate*, *moderate-high*, and *high*. According to the instrument manual, SAPROF is developed for use in conjunction with risk assessment instruments such as HCR-

20 in offender and forensic psychiatric samples. Research on samples other than the development samples has yielded inverse AUCs of as much as 0.85 (Abidin et al., 2013), but a recent meta-analysis did not support the conclusion that protective factors contribute to improve the accuracy of the summary judgment (O'Shea & Dickens, 2016).

In recent years, SAPROF has been supplemented with a youth version, SAPROF-YV (de Vries Robbé, Geers, Stapel, Hilterman, & de Vogel, 2015), and with self-appraisal versions for adults, SAPROF-ISA (de Vries Robbé & de Vogel, 2014) and youths, SAPROF-YV SA (de Vries Robbé & Hilterman, 2015).

1.6.5 Other instruments

A proliferation of violence risk assessment instruments has taken place over the last two to three decades; allegedly, there are now over 400 in the world (Singh et al., 2014), making an unabridged account impracticable. It may, however, be justified to mention a few of them, as they are among the most prominent and, in addition, represent different theories and needs. The semi-structured interview protocol *PCL-R*, Psychopathy Checklist-Revised (Hare, 1991) has been shown to correlate with violence risk (National Collaborating Centre for Mental Health, 2010), but it is actually intended for measuring psychopathy. *VRAG*, Violence Risk Appraisal Guide (Harris, Rice, & Quinsey, 1993), yielded a high degree of predictive accuracy (AUC = 0.76) for the development sample of apprehended males with mental disorders, but replications have produced lower values for other samples (Coid et al., 2009; Harris, Rice, & Camilleri, 2004; Hastings, Krishnan, Tangney, & Stuewig, 2011; Rossegger, Endrass, Gerth, & Singh, 2014). Being a purely actuarial instrument, VRAG does not allow for individual considerations. *START*, Short-Term Assessment of Risk and Treatability (Webster, Martin, Brink, Nicholls, & Desmarais, 2009), emanates from the structured clinical judgment approach and is designed to communicate risk of not only violence, but also self-harm and victimization within the next weeks to months. For each of its 20 items, strengths and vulnerabilities are coded independently. Meta-analytic data suggest a relatively strong ability to predict perpetration of interpersonal violence, AUC = 0.75 (O'Shea & Dickens, 2014).

1.7 VIOLENT VICTIMIZATION

Interpersonal violence always involves perpetration and victimization. In some respects, these two constructs are concomitant. For example, the conceptual definitions and typologies of violence may be useful in both cases. As for epidemiology and aetiology, there are both similarities and differences.

Rates of violent perpetration and violent victimization do correspond closely, and young males are overrepresented in each respect; females are, however, more often subjected to sexual violence and intimate partner violence (Daigle, 2012). Other risk factors include singleness, low socioeconomic status (Jennings & Reingle, 2014), and, interestingly, violent perpetration (Jennings, Piquero, & Reingle, 2012).

Mental disorders—severe mental disorders in particular—constitute another risk factor for violent victimization. Review studies have reported that annual victimization rates are 7.1-56% among those with such disorders (Latalova et al., 2014), corresponding to an at least twofold risk increase (Maniglio, 2009); and that there is a strong association between perpetration and victimization also in this population, $r = 0.50$ (Desmarais et al., 2014). Violence begets violence: victimization is in turn a risk factor for perpetration among forensic psychiatric patients (Sadeh, Binder, & McNiel, 2014) and individuals with psychotic disorders (Sariaslan, Lichtenstein, Larsson, & Fazel, 2016).

This association makes it hardly surprising that several risk factors are predictive of both perpetration and victimization, such as substance abuse, psychopathy (Silver, Piquero, Jennings, Piquero, & Leiber, 2011), and magnitude of psychiatric symptoms (Maniglio, 2009).

There appears to be a dearth of research dealing with protective factors for violent victimization among individuals with severe mental disorders. Studies on other samples demonstrate protective factors such as assertiveness (Schwartz, Dodge, & Coie, 1993), agreeableness (Egan & Perry, 1998), neighbourhood cohesion (Christiansen & Evans, 2005), school commitment, religiosity, verbal IQ (Daigle, Beaver, & Turner, 2010), and social support (Wenzel, Tucker, Elliott, Marshall, & Williamson, 2004). A related concept, resiliency, refers to the ability to recuperate from the detrimental repercussions of victimization (Dutton & Greene, 2010).

It is plausible that the health care services may contribute to the prevention of violent victimization. Such services might succeed in reducing risk factors like mental disorders, in facilitating protective factors like psychiatric treatment, and in bolstering resiliency from both psychological trauma and physical injuries.

1.8 PROJECT RATIONALE

This introduction has shown that violence is an almost ubiquitous health problem, that individuals with mental disorders are particularly exposed, and that violent perpetration and violent victimization are intertwined. Even though many facts and associations are established by previous research, several aspects remain unexplored. Methods for assessing violence risk must be evaluated in different contexts and across various populations, for the sake of both the individuals being assessed and the surrounding community, and the extent of these individuals' exposure to violence must be elucidated.

As offenders with mental disorders often exhibit relevant risk factors in terms of both psychiatric diagnoses and previous delinquency, they form a group where the risk of violence is particularly important to consider. This group is heterogeneous and displays a wide range of symptoms and criminal tendencies; furthermore, the group's composition changes from time to time and from place to place. Accordingly, there is a need for violence research in a Swedish forensic psychiatric context.

This thesis project aimed at validating different methods for assessing the risk of violent perpetration among offenders with mental disorders in Sweden, but also at surveying the prevalence of violent victimization in this group.

1.9 HYPOTHESES

The main hypotheses tested in this project were (1) that COVR, LSI-R, HCR-20^{V3}, SAPROF, and self-reported violent ideation could predict violent perpetration better than chance alone among offenders with mental disorders, (2) that violent ideation would be more common among offenders with mental disorders than general psychiatric patients, and (3) that violent victimization, health service utilization, and unmet health care needs would be more common among offenders with mental disorders than general population controls.

2 PROJECT SUMMARY

2.1 DESIGN

This doctoral thesis project comprised three studies.

Study I, *Violence during imprisonment, forensic psychiatric care, and probation: Correlations and predictive validity of the risk assessment instruments COVR, LSI-R, HCR-20^{V3}, and SAPROF*, was a naturalistic, prospective cohort study in which four risk assessment methods were validated in a forensic psychiatric sample.

Study II, *Violent victimization and health service utilization in a forensic psychiatric context: a comparison between offenders with mental disorders and matched controls*, was a descriptive study, were self-reported rates of violent victimization and health service utilization were compared between a forensic psychiatric sample and a sample of matched controls from the general population.

Study III, *Self-reported violent ideation and its link to interpersonal violence among offenders with mental disorders and general psychiatric patients*, was also a naturalistic, prospective cohort study in which rates of violent ideation and its association with subsequent violent acts were reported for a forensic psychiatric sample and a general psychiatric sample.

2.2 SAMPLES

2.2.1 Forensic psychiatric sample

The forensic psychiatric sample consisted of 200 detained persons undergoing a forensic psychiatric evaluation at an assessment unit in Stockholm during the years 2011 to 2013. Study participants were required to be between 16 and 60 years old.

In order to characterize this sample, the forensic psychiatric system in Sweden must be described. Forensic psychiatric evaluations are conducted at the request of the court, to find out whether suspects of crimes for which the sanctions cannot be limited to a fine (1) suffer from a severe mental disorder, (2) committed the crime under the influence of a severe mental disorder, (3) are in need of compulsory care, and (4) run the risk of relapsing into serious crimes due to the mental disorder (Swedish Penal Code, 1962).

In Sweden, severe mental disorder is a legal rather than medical term; it comprises conditions such as psychoses, major depression with suicidal ideation, severe personality disorders with psychotic episodes, and in some cases also severe dementia, severe brain damage, or profound intellectual disabilities (Government bill 1990/91:58, 1990). Swedish law does not allow acquittal of perpetrators by reason of insanity; offenders suffering from mental disorders are sentenced to forensic psychiatric care.

2.2.2 General population control sample

The control sample consisted of 600 persons from the general population. For each person in the forensic psychiatric sample, there were three controls matched by age span (16-25, 26-35, 36-45, 46-55, and 56-65 years), sex, and occupation (employed, unemployed, student).

2.2.3 General psychiatric sample

The general psychiatric sample consisted of 390 patients recruited at discharge from two public psychiatric hospitals in Stockholm during the year 2007. Study participants were required to be between 16 and 60 years old and able to take part in interviews in Swedish or English, and to have a Swedish social security number and an ICD diagnosis.

2.3 MEASURES

2.3.1 Baseline data

2.3.1.1 Sample characteristics

Demographic, clinical, and criminal data were used to describe the sample characteristics. These data comprised age, sex, marital status, occupation, country of birth, diagnosis, occurrence of severe mental disorder, criminal history, current offence, and sanction.

2.3.1.2 Violence risk

Four instruments were used to assess the risk of future violence in study I. These instruments are more thoroughly described in the introduction.

COVR, Classification of Violence Risk (Monahan et al., 2005), is an actuarial instrument designed to assess the risk of violence among general psychiatric patients within the next months. This method is based on a structured interview, where the rater reads questions from a computer screen. Due to a classification tree design, different interviews do not always comprise the same set of questions. At the end of the interview, the software generates a report in which the risk is described as *very low*, *low*, *average*, *high*, or *very high*.

LSI-R, Level of Service Inventory-Revised (Andrews and Bonta, 1995b), is also a predominantly actuarial instrument. Among offenders, it is intended to identify both needs and risks regarding criminal recidivism, not necessarily violent recidivism. The instrument comprises 54 items covering different criminogenic factors; high scores reflect a high propensity of reoffending.

HCR-20^{V3}, Historical Clinical Risk Management-20, version 3 (K. S. Douglas et al., 2013), is a structured professional judgment instrument, developed to assess the risk of future violence in both forensic psychiatric and correctional practice. Its 20 items cover past, current, and future factors; additional items may be added in the individual case. In this project, both risk categories and total scores were considered. The risk categories—*low*, *moderate*, and *high*—were determined by considering the relevance of each item; this means that a small number

of items of high relevance may entail a high risk and vice versa. The total score, ranging from 0 to 40, was calculated by assigning each item the values *0* (not present), *1* (partially or possibly present), or *2* (present).

SAPROF, Structured Assessment of Protective Factors (de Vogel et al., 2009), is another structured professional judgment instrument. Though it is designed to assess violence risk, it is based on protective factors rather than risk factors. It comprises 17 items, covering internal, motivational, and external factors. Protection categories (*low*, *moderate*, *high*) and total scores (0-34) were considered; they were determined in the same way as for HCR-20^{V3}. It may be worth clarifying that a low protection category and a low score both represent a high risk.

HCR-20^{V3} and SAPROF scores were also combined by subtracting the latter from the former. The rationale is that SAPROF is intended to be used in conjunction with HCR-20 or related tools; the subtraction procedure is also described in the SAPROF manual.

2.3.1.3 Violent victimization

In study II, the variable concerning self-reported violent victimization during the past twelve months was divided into four categories: violence that occasioned health care, violence that caused perceptible injuries, violence that did not cause perceptible injuries, and threats causing fear. This variable was taken from a questionnaire used by the governmental agency Statistics Sweden to conduct the Swedish Living Condition Surveys, SILC. These surveys, conducted since 1975, cover areas such as health, employment, and security.

2.3.1.4 Health care utilization

To measure different aspects of health service utilization in study II, two variables were used. Health service utilization per se was defined as self-reported doctor's appointment during the past three months because of own illness. Unmet health care needs were defined as self-reported unmet health care needs during the past three to twelve months. These two variables were also taken from the SILC questionnaire.

2.3.1.5 Violent ideation

Violent ideation, addressed in study III, was defined as self-reported daydreams or thoughts about physically hurting or injuring other persons. Depending on its temporal proximity, the variable was divided into two: violent ideation ever and violent ideation during the past two months. Both the definition and the division emanate from two items in the COVR instrument.

2.3.2 Follow-up variables

Perpetration of interpersonal violent acts constituted the outcome variable in studies I and III. This variable was conceptualized according to the definition of the World Health Organization, i.e. the threatened or actual intentional use of physical force of power against

another person that either results in or has a high likelihood of resulting in injury, death, psychological harm, maldevelopment, or deprivation (World Health Organization, 2002). The operationalization of interpersonal violent acts varied across samples and studies because of different sources of follow-up data: records, crime conviction registry, or interviews.

2.4 PROCEDURE

2.4.1 Baseline procedure

For the forensic psychiatric sample, data were gathered from interviews and case files. Data gathering took place at an assessment unit in Stockholm in conjunction with court ordered forensic psychiatric evaluations before sentence. Demographic, clinical, and criminal information was obtained. The instruments COVR, LSI-R, HCR-20^{V3}, and SAPROF were used to assess the risk of future violence for study I. For study II, a section of the SILC questionnaire was used regarding previous victimization and health service utilization. Finally, two COVR items were used to gather information about violent ideation for study III.

For the general population control sample of study II, baseline data were gathered from telephone interviews; participants could reside anywhere in society and be engaged in any activity. The interviews were conducted by Statistics Sweden, a government agency, as part of its regular SILC surveys. Demographic information was obtained, and participants were asked questions about previous victimization and health service utilization.

For the general psychiatric sample of study III, demographic, clinical, and criminal baseline data were gathered from interviews and records at discharge from psychiatric hospitals. To get information about violent ideation, two items from the COVR instrument were used.

2.4.2 Follow-up procedure

The forensic psychiatric sample was followed up regarding violent perpetration after 20 weeks in study III and after 52 weeks in study I. At the first occasion, information was gathered from the crime conviction registry and from records, and at the second occasion from records. Depending on their sanctions, participants could reside at different sites at follow-up; some at forensic psychiatric hospitals and some at prisons, and a few in society. The general population control sample was not followed up, as study II was not prospective. The general psychiatric sample of study III was also followed up regarding violent perpetration, but only once. After 20 weeks, information about violent perpetrations was gathered from the crime conviction registry and from follow-up interviews with participants and associates. At follow-up, participants could reside anywhere in society.

2.4.3 Statistical analyses

To compare groups, χ^2 tests and Fisher's exact tests were used for categorical data, and Mann-Whitney *U* tests and ANOVAs for continuous data. Cohen's κ and ICC(2,1) coefficients were computed to assess interrater reliability; and Spearman and Pearson coefficients to determine correlations. Predictive validity was evaluated by means of ROC

curves, AUCs, sensitivity, specificity, positive and negative predictive values, and φ coefficients. Finally, logistic regressions were carried out to obtain risk ratios and odds ratios. The statistical methods of the thesis are described in further detail in the appendix.

2.5 RESULTS

2.5.1 Sample characteristics

Characteristics of the samples are described in Table 6. On average, participants were in their thirties. In the forensic psychiatric sample, a majority were males (87.0%), psychosis was the most common main diagnosis (28.5%), and rates of previous violence were high (92.5%).

Table 6. Sample characteristics.

	Forensic psychiatric sample (study I, II, III) *N* = 200	**General psychiatric sample (study III)** *N* = 390[a]	**General population controls (study II)** *N* = 600
Age	31[b]	37[c]	31[b]
Sex			
Male	174 (87.0%)	187 (47.9%)	522 (87.0%)
Female	26 (13.0%)	203 (52.1%)	78 (13.0%)
Single	146 (73.0%)	-	-
Occupation			
Employed	32 (16.0%)	-	96 (16.0%)
Unemployed	156 (78.0%)	-	468 (78.0%)
Student	12 (6.0%)	-	36 (6.0%)
Foreign born	75 (37.5%)	105 (26.9%)	-
Mental disorder			
Severe[d]	93 (46.5%)	-	-
Mood disorder[e]	11 (5.5%)	109 (32.9%)	-
Psychosis[e]	57 (28.5%)	58 (17.5%)	-
Substance use disorder[e]	31 (15.5%)	20 (6.0%)	-
Personality disorder[e]	31 (15.5%)	49 (14.8%)	
Violence			
Previous	185 (92.5%)	37 (10.0%)	-
Current offence	181 (90.1%)	-	-
Sanction			
Forensic psychiatric care	88 (44.0%)	-	-
Prison[f]	82 (41.0%)	-	-
Probation	27 (13.5%)	-	-
Dismissed	3 (1.5%)	-	-

Note. [a]369 for previous violence, 331 for diagnoses. [b]Median age in years. [c]Mean age in years. [d]Severe in Swedish legal sense. [e]Main diagnosis. [f]Including one case where prison was combined with probation.

Because of the matching, the general population control sample was commensurate with the forensic psychiatric sample regarding age span, sex, and occupation. The general psychiatric sample had an even gender distribution, mood disorders constituted the largest diagnostic group (32.9%), and previous violence was fairly infrequent (10.0%).

2.5.2 Rates of violent perpetration, violent victimization, health service utilization, and violent ideation

Details of rates of violent perpetration, violent victimization, health service utilization, and violent ideation are provided in Table 7.

Table 7. Rates of violent perpetration, violent victimization, health service utilization, and violent ideation.

	Forensic psychiatric sample (Study I, II, III)			**General psychiatric sample (Study III)**	**General pop. controls (Study II)**
	Entire	SMD+[a]	SMD-[b]		
	N = 200[c]	*n* = 92[d]	*n* = 108[e]	*N* = 390[f]	*N* = 600[g]
Perpetration[h]					
Violence 20w	16 (8.2%)	-	-	35 (11.8%)	-
Violence 52w	45 (23.3%)	30 (33.7%)	15 (14.4%)	-	-
Threats 52w	35 (18.1%)	25 (28.1%)	10 (9.6%)	-	-
Victimization[i]					
Any	104 (52.3%)	40 (44.0%)	64 (59.3%)	-	65 (11.1%)
Violence	86 (43.2%)	29 (31.9%)	57 (52.8%)	-	41 (7.0%)
Utilization[j]					
Appointment	95 (47.7%)	42 (46.2%)	53 (49.1%)	-	142 (23.7%)
Unmet need	84 (42.2%)	35 (38.5%)	49 (45.4%)	-	100 (16.7%)
Ideation[k]					
Ever	65 (32.5%)	-	-	139 (35.7%)	-
Recent	45 (22.5%)	-	-	81 (20.8%)	-

Note. [a]Forensic psychiatric sample with severe mental disorders. [b]Forensic psychiatric sample without severe mental disorders. [c]195 for perpetration within 20 weeks, 193 for perpetration within 52 weeks, 199 for victimization and utilization. [d]89 for perpetration, 91 for victimization and utilization. [e]104 for perpetration. [f]296 for perpetration, 389 for ideation. [g]588 for victimization, 599 for appointment, 598 for unmet need. [h]Perpetration of violence or threats within 20 or 52 weeks after baseline. [i]Subjected to threats or violence, or to violence only, during 52 weeks before baseline. [j]Health service utilization during 3 months before baseline. [k]Violent ideation before baseline, ever or past 2 months.

Violent perpetration rates are reported in studies I and III. During 20 weeks after baseline, violent perpetration was slightly more frequent in the general psychiatric sample (11.8%) than in the forensic psychiatric sample (8.2%). Within the latter sample, violent perpetration

occurred about twice as often during 52 weeks after baseline among those with severe mental disorders (33.7%) compared to those without such disorders (14.4%).

As described in study II, violent victimization during 52 weeks before baseline was reported by 52.3% of the forensic psychiatric sample and by 11.1% of the matched controls from the general population (*RR* = 8.2). Forensic psychiatric participants with severe mental disorders reported violent victimization less often than those without such disorders, 44.0% vs 59.3%; and those with unmet health care needs more often than those without such needs, 61.9% vs 45.2%.

Health service utilization rates are also accounted for in study II. Doctor's appointments during three months before baseline were reported by 47.7% of the forensic psychiatric sample—the proportion was approximately the same regardless of presence of severe mental disorder—and by 23.7% of the general population controls (*RR* = 2.0). Unmet health care needs were also reported more often in the forensic psychiatric group (42.2%) than in the control group (16.7%), corresponding to a risk ratio of 3.4.

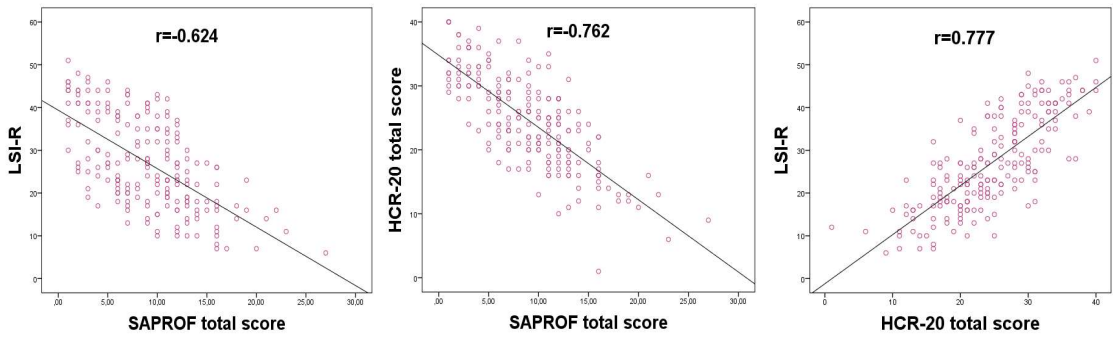
Violent ideation rates, dealt with in study III, were similar across the forensic psychiatric sample and the general psychiatric sample. Slightly less than a quarter reported that they had experienced violent ideation during two months before baseline (22.5% vs 20.8%), and about a third that they had ever done so (32.5% vs 35.7%).

2.5.3 Violence risk prediction

Violence risk prediction performances, calculated in studies I and III, were reported in different ways, including correlations and AUCs.

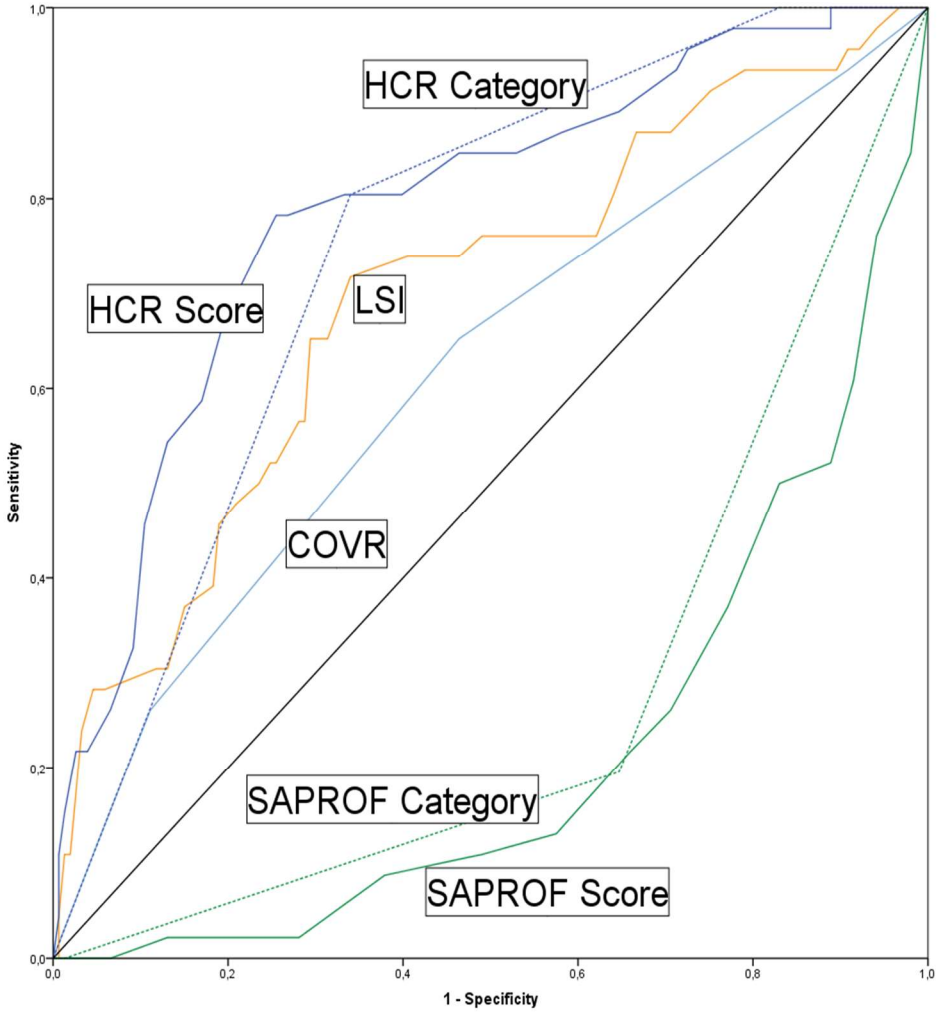
In study I, the correlations between the risk assessment instruments were calculated. Pearson correlations regarding the total scores of LSI-R, HCR-20^{V3}, and SAPROF, are displayed in Figure 3. As for absolute values, coefficients were around 0.70, indicating considerable correlations. Spearman correlations between the risk categories of COVR, LSI-R (LSI-R score was treated as categorical data), HCR-20^{V3}, and SAPROF, were more dispersed, in absolute values ranging from 0.30 to 0.64.

Figure 3. Correlations between total scores of LSI-R, HCR-20^{V3}, and SAPROF.



ROC curves, illustrating the association between risk estimates and violent perpetration within 52 weeks in the forensic psychiatric sample, are displayed in Figure 4. Guidelines provided by Rice & Harris (1995) were used to interpret AUCs. In study I, AUCs were small for COVR (0.61), medium for LSI-R (0.70), and large for HCR-20^{V3} score (0.79) and summary rating (0.74), for SAPROF score (0.78) and summary rating (0.73), and for the difference between HCR-20^{V3} and SAPROF scores (0.81). In study III, there appeared to be an association between violent ideation ever and violent acts when both samples were combined, *OR* = 2.42, 95% CI [1.35, 4.36]; but for the forensic psychiatric sample, AUCs were below small for violent ideation during past two months (0.51) and ever (0.52). In the general psychiatric sample, the predictive validity was small for violent ideation during past two months (AUC = 0.57) and medium for violent ideation ever (AUC = 0.65).

Figure 4. ROC curves for COVR, LSI-R, HCR-20^{V3}, and SAPROF; SAPROF curves are not inverse.



3 DISCUSSION

3.1 GENERAL FINDINGS

In line with the first hypothesis, the results of study I corroborate the notion that COVR, LSI-R, HCR-20^{V3}, and SAPROF constitute methods suitable for predicting violent perpetration within a year among offenders with mental disorders on a group level. Correlations between the scores of the instruments were considerable, between the summary ratings somewhat poorer. Even so, the instruments were not equivalent in accuracy. For COVR, the predictive performance was small (AUC = 0.61); it is true that previous research has demonstrated a fair validity in forensic psychiatric settings (Snowden et al., 2009), but the instrument was originally developed in a general psychiatric context (Monahan et al., 2006). Consistent with previous research (de Vogel et al., 2009; Singh, Grann, et al., 2011), the predictive performance was medium for LSI-R (AUC = 0.70), large for HCR-20^{V3} (AUC = 0.79), and large for SAPROF (AUC = 0.78). Although a body of other studies have established the association between violent ideation and violent behaviour (Bjerrum Moeller et al., 2017; Grisso et al., 2000; Nagtegaal et al., 2006), violent ideation performed less well as a predictor of violence in study III (AUC = 0.52). Comparisons across studies are, however, deceptive, as there are several performance indicators measuring different facets of predictive validity (Singh, Desmarais, & Van Dorn, 2013). Violent ideation should not be ruled out as a risk factor, but additional factors must be taken into account when formulating a risk in the individual case.

The second hypothesis was not supported; in study III, self-reported violent ideation did not seem to be more common among offenders with mental disorders than general psychiatric patients. These results are not entirely in line with previous research. As for violent ideation during the past two months, for example, a rate of 46.0% has been reported for a forensic psychiatric sample (Bjerrum Moeller et al., 2017) and 29.8% for a general psychiatric sample (Grisso et al., 2000); in study III, the corresponding rates were 22.5% and 20.8%, respectively. A possible explanation for this discrepancy is that the make-up of samples varies between countries because of different demographics and legislations. The samples of study III did not differ regarding violent acts during the follow-up time either, but this comparison is unreliable because of considerably disparate settings. It cannot be precluded that mediating mechanisms such as anticipation, appraisal, and control may vary between the samples, so that violent ideation causes different repercussions.

Finally, the results lend some support to the third hypothesis, dealt with in study II. Violent victimization, health service utilization, and unmet health care needs were reported more often by offenders with mental disorders than general population controls. Among the offenders, 53.2% reported violent victimization and 47.7% recent health service utilization, fairly high numbers in comparison with other psychiatric samples, for which victimization rates from 6.4% to 56.0% (Latalova et al., 2014) and utilization rates from 25.7% to 52.0% (Fleury, Grenier, Bamvita, Perreault, & Caron, 2012; Wang et al., 2005; Wittchen & Jacobi,

2005) have been reported. Even though there are differences regarding conceptualizations and time frames, these comparisons indicate that offenders with mental disorders are more vulnerable than other individuals with such disorders. In study II, offenders with severe mental disorders were victimized less often (44.0%) than offenders with other mental disorders (59.3%). A possible explanation could be that the former group receives more professional interventions that reduce the exposure; there were, however, no significant differences between the groups regarding health service utilization (46.2% vs 49.1%). The importance of professional interventions is nevertheless indicated by the association between unmet health care needs and violent victimization.

3.2 METHODOLOGICAL CONSIDERATIONS

3.2.1 Sampling and matching

Appropriate sampling is a prerequisite for drawing valid conclusions about a population. In this project, non-probability sampling methods were employed. Generally, probability sampling methods are preferable (D. Bowers, House, & Owens, 2011), as they can protect against sampling bias. There were indeed some differences between participants and non-participants in the projects. For example, forensic psychiatric participants suffered less often from severe mental disorders than non-participants, and as severe mental disorders seemed to correlate with violent outcomes, the sample may not have been entirely representative in this respect. However, those who are asked to participate must, of course, have the right to say no.

Consecutive sampling was employed to enrol the forensic psychiatric sample of studies I, II, and III. This non-probability method (Martínez-Mesa, González-Chica, Duquia, Bonamigo, & Bastos, 2016) implies that every person who meets the inclusion criteria is approached until the requisite sample size is attained. Thus, the method serves to reduce sampling bias associated with regular convenience sampling. As the enrolment period was two entire years, the risk of possible seasonal influences was probably negligible (Schuster & Powers, 2005).

To obtain the general psychiatric sample of study III, convenience sampling was applied; only reasonably accessible patients were approached at discharge. The method was chosen for practical reasons; it would not have been affordable to have research assistants monitoring the hospitals round-the-clock. It is conceivable that patients discharged at inconvenient hours may me more impulsive and less motivated, and hence more prone to violence; but on the other hand, most discharges probably take place during office hours, or at least in the daytime.

The general population sample of study II consisted of controls matched with forensic psychiatric participants on age span, sex, and occupation. Matching aims at eliminating confounding by the matching factors (de Graaf, Jager, Zoccali, & Dekker, 2011). An alternative would be to adjust for these factors in a regression model (Stuart, 2010), but matching may be preferable if they are expected to differ drastically between groups (Rose & Laan, 2009), as was the case in this project. A disadvantage of matching is that it is not possible to study the impact of the matching factors on the outcome variable (Katz, 2006), but

in this case, their relationship is already established. Another disadvantage is that matching makes sample and controls more similar not only regarding matching factors, but also explanatory variables (Pearce, 2016); hence, the number of matching factors was restricted. There are several different matching methods, and one of the most common is based on propensity scores. In this project, the propensity score would be the probability that a person with certain baseline characteristics (age span, sex, occupation) will belong to the forensic psychiatric sample as opposed to the control group. The most common method to estimate propensity scores is to use a logistic regression model in which group affiliation is regressed on these baseline characteristics (Austin, 2011a). Forensic psychiatric participants and controls are then matched on propensity scores (Austin, 2011b). In this sense, propensity score matching simulates a complete randomization design (Okoli, Sanders, & Myles, 2014). However, the matching method used in this project mimics a blocking design (King & Nielsen, 2016), which may reduce variability (e.g. between males and females) and thus improve precision (N. H. Anderson, 2001).

3.2.2 Measures and procedure

When gathering data, the researcher must pay attention to the risk of obtrusive measurement; that is the risk that the measuring affects the outcome. An example from the field of physics may illustrate the concept: when a thermometer is inserted in liquid, the procedure may alter the liquid's temperature because of friction and heat exchange (Bunge, 1985). In the field of violence risk assessment, obtrusive measurement may occur if the estimated risk results in interventions which reduce the risk (K. S. Douglas & Kropp, 2002). The prospective studies I and III in this thesis project had an unobtrusive design, so the baseline ratings were not conveyed to the forensic psychiatric hospitals, prisons, or probation offices. However, these authorities most probably carried out their own assessments, thus altering the outcome.

As outlined in the introduction, there is a lack of consensus as to operationalizing and measuring violence. Hence, there is always a risk that the measuring method does not capture all the violent events it is supposed to. A way to overcome, or at least reduce, this problem is data triangulation, that is the use of multiple sources of data in an investigation (Salkind, 2010). This method was employed in study III, in which both records and registries provided data on violent perpetration. The latter source was far less comprehensive, thus demonstrating the logic behind triangulation.

3.2.3 Correlations

In this project, Spearman and Pearson coefficients were calculated to measure correlations between the risk assessment instruments in study I. The Spearman statistic assumes that data are ordinal, e.g. summary ratings, whereas the Pearson statistic assumes continuous data, e.g. total scores. Some authors contend that scores also constitute ordinal data (Koch, 2015) as steps on a scale are not necessarily equidistantly spaced; if so, the Spearman statistic should be used for scores too.

3.2.4 Dropouts

The number of dropouts may have posed a risk of impaired inferences, especially in study III in which 28.1% of the general psychiatric sample was lost to follow-up. The approach that was chosen to handle these missing data, complete case analysis, reduces sample size and may increase the potential of bias. For this reason, a multiple imputation approach was employed as well. The model included the variables sex, age, and previous conviction of violent crime, and conducted the imputation five times. As for the results, the differences between the two approaches were negligible. However, it is possible that data were not missing at random. It may have been that the least violent forensic psychiatric participants and the most violent general psychiatric participants were lost to follow-up; the former because they were discharged after relatively short lengths of stay and the latter because of turbulent life conditions. If data are not missing at random, multiple imputation should be avoided (Sterne et al., 2009). Sensitivity analysis may be an option, but routine use of this method in applied research is lacking (Smuk, Carpenter, & Morris, 2017). Thus, the choice of complete case analysis may be warranted.

3.2.5 Validation

When evaluating risk assessment methods, as in studies I and III, validity is an important concept. It refers to the degree to which an instrument actually measures what it purports to measure (Sim & Wright, 2000). The concept was minted at least 100 years ago and comprises several approaches (Lissitz & Samuelsen, 2007), accounted for in Table 8. The cornerstone is the construct validity, which ultimately measures how well an operational definition reflects the conceptual definition (Schachter, Gilbert, Wegner, & Hood, 2016).

Table 8. Validity and reliability.

Concept	The extent to which…
Research validity	
Internal validity	independent and dependent variables are causally associated
External validity	the results of a study can be extrapolated
Ecological validity	the results of a study can be extrapolated in real life
Test validity	
Construct validity	a test measures what it is supposed to measure
Content validity	a test measures all aspects of a construct
Face validity	a test seems to measure all aspects of a construct
Criterion validity	a test measures an outcome
Concurrent validity	a test measures a concurrent outcome
Predictive validity	a test measures a future outcome
Reliability	
Alternate-form reliability	scores of different test versions are consistent
Internal consistency	subscales or items are consistent with the entire test
Interrater reliability	test results are consistent between raters
Test-retest reliability	test results are consistent between occasions

How well different methods perform is one of the key issues in research of prediction. Quite easy to comprehend are the concepts *sensitivity* (the probability that a violent person is classified as violent), and *specificity* (the probability that a non-violent person is classified as non-violent); both of which have been used to measure validity (Sheldon, Davies, & Howells, 2011). These two measures reflect the intrinsic characteristics of a test (Maxim, Niebo, & Utell, 2014).

ROC curves incorporate sensitivity and specificity, displaying the true positive rate (sensitivity) on the vertical axis and the false positive rate (1 - specificity) on the horizontal axis for different cut-offs of the instrument (Florkowski, 2008). Developed during the Second World War, the method was originally designed to differentiate radar signals from noise; ROC stands for receiver operating characteristics (Fan, Upadhye, & Worster, 2006). The area under the ROC curve, *AUC*, is an often reported effect size; the maximum possible AUC of an ideal instrument is 1, whereas an AUC of 0.5 indicates that the instrument does not predict an outcome such as violence better than chance. Important to bear in mind is that an instrument suited for identifying low-risk individuals may have the same AUC as one suited for identifying high-risk individuals (Fazel & Bjørkly, 2016). The maximum height between the ROC curve and the chance line may be used to determine the proper cut-off score of the instrument; this height corresponds to the maximum *Youden index*, that is sensitivity + specificity - 1 (Kattan & Cowen, 2009). To conclude, AUC has become the standard measure of validity in research of violence risk assessment (Singh et al., 2013), and was hence the measure of choice in this project.

The *positive predictive value* (the probability that a person classified as violent is violent) and *negative predictive value* (the probability that a person classified as non-violent is non-violent) are also measures of validity (Sheldon et al., 2011). These are considerably affected by the prevalence of violence in the group being assessed. If, for example, sensitivity amounts to 80%, specificity to 90%, and prevalence to 50%, then Bayes's theorem gives a positive predictive value of 88%; but if the prevalence is 1%, the positive predictive value is only 7.5% (Riegelman, 2013). To a clinician, predictive values are probably more interesting than sensitivity and specificity; the classification should precede the outcome and not the other way round. Thus they were reported in the clinically oriented study III.

Alternative ways to report validity could have been used in this project. Likelihood ratios combine information from both sensitivity and specificity. *The positive likelihood ratio* is the probability of a violent person being classified as violent (true positive rate or sensitivity) divided by the probability of a non-violent person being classified as violent (false positive rate or 1 - specificity); if instead the probability of a violent person being classified as non-violent (false negative rate or 1 - sensitivity) is divided by the probability of a non-violent person being classified as non-violent (true negative rate or specificity), the *negative likelihood ratio* is obtained (Riegelman, 2013). A positive ratio (which can vary from 1 to infinity) above 5 and a negative ratio (which can vary from 1 to 0) below 0.3 suggest that the instrument is relatively accurate (National Collaborating Centre for Mental Health, 2015).

The positive likelihood ratio divided by the negative gives the *diagnostic odds ratio*, ranging from zero to infinity with higher values indicating better discriminatory performance of the test (Glas, Lijmer, Prins, Bonsel, & Bossuyt, 2003). These three ratios are relatively independent of prevalence. *Cohen's d* is an effect size for the difference in mean scores between a violent and a non-violent group, expressed in standard deviations (Rice & Harris, 1995). According to Cohen, 0.2 represents a small, 0.5 a moderate, and 0.8 a large effect (J. Cohen, 1992). A supplementary rule of thumb has been suggested, in which 0.01 represents a very small, 1.2 a very large, and 2.0 a huge effect (Sawilowsky, 2009). The *common language effect size* refers to the probability that a randomly selected score from the violent group is higher than a randomly selected score from the non-violent group (McGraw & Wong, 1992; Rice & Harris, 1995). *Point-biserial correlations* may be used when the independent variable is continuous or ordinal, for example risk scores or categories, and the dependent variable is dichotomous, such as violence. In general, *r* values of 0.1, 0.3, and 0.5 are regarded as small, medium, and large effects, respectively, (J. Cohen, 1988). However, these values assume a base rate of 50%, perhaps not typical of violence.

3.2.6 Reliability

Reliability may be described as the stability of an instrument in different settings (Franzen, 2000). There are four main types of the concept: alternate-form reliability, internal consistency, interrater reliability, and test-retest reliability (Fink, 1995), explained in Table 8. Research on violence risk assessments typically deals with interrater reliability, that is the agreement among raters. In this project, interrater reliability was measured for the risk assessment instruments in study I.

There are several ways to calculate reliability for categorical data. *Percent agreement*, i.e. the number of agreement scores divided by the total number of scores, is easy to grasp and interpret. It does not, however, take chance agreement into consideration, but *Cohen's* κ does. The κ statistic is based on a χ^2 table and takes observed and expected agreement into account. In theory, κ ranges from -1 to 1, in practice rather from 0 to 1, where higher values reflect higher agreement. Cohen's κ has some limitations; it cannot, for example, be directly interpreted, and it assumes independence between raters (McHugh, 2012). In study I, κ coefficients were calculated for measuring the agreement for the summary ratings of HCR-20^{V3} and SAPROF. Coefficients were not weighted, but that could have been an option; disagreements may have been scaled, as summary ratings—low, moderate, high—are ordinal rather than nominal. Other agreement indices for ordinal data are the *Kendall correlation coefficients* or the *Spearman rank correlation coefficient*, but the latter method does not account for systematic bias (Jakobsson & Westergren, 2005).

Intraclass correlation was calculated to measure reliability for continuous data, that is total scores of LSI-R, HCR-20^{V3}, and SAPROF. Ranging from 0 to 1 it may still be difficult to interpret; it is a complicated measure that exists in different forms (Koo & Li, 2016). *Pearson correlations* may seem a more fathomable alternative; but in this case, correlation is measured rather than agreement. Suppose, for example, that rater a scores are consistently 3

points higher than rater b scores; than there would be a total positive correlation but not a total agreement.

3.3 LIMITATIONS

The external validity of the project may have been hampered for the reason that the Swedish legal system has greatly affected the make-up of the forensic psychiatric sample; the results may not reflect conditions in other countries. On the other hand, if studies from different countries are combined, for example in a meta-analysis, external validity will ultimately improve.

Although sufficiently large to conduct conclusive validation analyses, the size of the forensic psychiatric sample ($N = 200$) did not allow for comparisons between all relevant subgroups. It is true that the mean sample size of violence risk assessment studies is larger ($N = 296$), but the use of larger samples does not necessarily improve predictive validity (Singh, Grann, et al., 2011).

Different types of bias may have affected the results. *Participation bias* may have led to an underestimation of the rate of violent perpetration in studies I and III; severe mental disorders seemed to be associated with violent acts, and non-participants suffered more often from such disorders. There were, however, no significant differences with regard to other demographic and criminological variables. As mentioned, *sampling bias* may have occurred due to the convenience sampling of general psychiatric patients in study III. In all studies, many data originated from the participants themselves, thus entailing a risk for *cognitive bias*; but corroborating case file information was gathered when possible. Especially with regard to the structured professional judgment instruments in study I, there may have been a risk of *rater bias* if those being assessed appeared unprepossessing; but raters were professional, and the aforementioned instruments correlated significantly with actuarial instruments. *Attrition bias* may have arisen from the exclusion of dropouts from analyses of follow-up data in studies I and III, but only a few offenders were lost to follow-up. As the aetiology of violence is complex and to a large extent unknown, the results in all studies may have been affected by *confounding bias*; but the project did not aim primarily at determining causal associations. The choice of AUC as the only performance indicator for the assessment instruments in study I may have posed a risk of *confirmation bias*, as large AUCs do not automatically bring about large predictive values; but this risk may have been reduced because of the high prevalence of violent perpetration in the sample in question. There was a potential *detection bias* in study III as offenders and patients were not followed up in an entirely consistent way, but the main purpose of the study was not to compare the follow-up variable between samples.

One important limitation is that violent perpetration, that is the dependent variable in study I and study III, was probably heavily influenced by the management of risk. In other words, accurate predictions may have been "ruined" by preventive measures. This effect has been illustrated in a follow-up study of spousal assault cases, in which higher levels of intervention

were associated with decreased recidivism in high risk cases, but also with increased recidivism in low risk cases (Belfrage et al., 2012).

3.4 ETHICAL CONSIDERATIONS

Autonomy, beneficence, non-maleficence, and justice form the *four principles approach* (Beauchamp & Childress, 1979), widely used in the field of medical ethics (Page, 2012). Although slightly differently phrased, these principles imbue the ethical standards for psychiatric practice, the Hawaii and Madrid declarations (Blomquist, 1978; Helmchen & Okasha, 2000).

To exercise respect for *autonomy*, the patient must be given the right to make deliberate decisions. In this project, participants gave their consent to undergo violence risk assessments, but that is not always the case in forensic and clinical contexts. For example, a patient who refuses to take part in a court-ordered assessment will probably be assessed anyway. Lack of empowerment may, however, make patients more reluctant to disclose important information; hence, the accuracy of the estimation may be impaired.

Beneficence entails that psychiatrists must be guided primarily by concern for the welfare and integrity of patients. A violence risk assessment may indeed be in the interest of the patient; it may identify beneficial treatments and facilitate earlier discharge, but even when it results in a prolonged length of stay, it may prevent reoffending and hence future detention (T. Douglas, Pugh, Singh, Savulescu, & Fazel, 2017). Some scholars contend that risk assessments should be carried out only if benefits will result (Buchanan & Grounds, 2011).

The third principle, *non-maleficence*, may be seen as a corollary of beneficence. Even the ancient Hippocratic Oath stressed that the physician must abstain from doing anything deleterious to the patient (S. H. Miles, 2004). It is possible, though, that mental health professionals using violence risk assessment instruments violate this principle. Among those whose risk is assessed as high, the false positive rate may be over 50% (Fazel, Singh, Doll, & Grann, 2012); as was consistently the case in study I in this project. However, this does not necessarily imply inaccurate assessments; measures could have been taken to prevent the predicted violence from occurring. Nevertheless, some patients are unjustly deprived of liberty. On the other hand, psychiatrists must balance professional obligations to the patients with responsibilities for the common good (Okasha, 2003).

Justice has been described as fairness; thus, the concept may denote fair distribution of resources, respect for human rights, and legal justice (Gillon, 1994). Violence risk assessments may bring about detention—the scope of which is often unforeseeable for the patient—because of acts not yet committed. This may seem incompatible with the legal principles *nullum crimen sine lege* (no crime without law) and *nulla poena sine lege* (no penalty without law); hence, it is important that detention not be the only risk management strategy. Veracity is also imperative; the patient must be informed of why an assessment is required, of its result, and of what actions it gives rise to.

3.5 IMPLICATIONS

This project had a naturalistic design, so the number of exclusion criteria was restricted. A consequence could have been heterogeneous samples with an increased risk of confounding, but this may also have improved generalizability (Humphreys, 2017). But how useful is this generalizability for clinicians? Quantitative studies typically imply that sample data are used to draw conclusions about a population rather than an individual (Rothwell, 2007), and if conclusions about individuals are based on group data, there is a risk of ecological fallacy. The problem is that the individual aspect cannot be disregarded in clinical practice; "Anyone who believes that anything can be suited to everyone is a great fool, because medicine is practiced not on mankind in general, but on every individual in particular", as French surgeon Henri de Mondeville put it as early as the 1310s (McDonald, 2004). For example, suppose that a COVR assessment indicates that the risk of violence for a certain patient is 56%. What measures should the estimator take? Clearly, this information does not suffice. And even if the risk was 100%, questions would still multiply. In what situation would the violent act take place? When? How? Against whom?

A possible way forward may be to create an individual risk formulation, in which these questions are addressed. Four major approaches to violence risk formulation have been developed (Hart & Logan, 2011). In the *offence paralleling behaviour* approach, the formulation includes a systematic analysis to identify antecedents and reinforcers of delinquency and other behaviour serving the same function (Daffern, Jones, & Shine, 2010). The *good lives model* approach promotes a formulation of socially acceptable goals, in which risk factors are viewed as obstacles to acquire these goals (Ward & Maruna, 2007). Risk formulations according to the *risk-needs-responsivity* approach convey not only the risk per se, but also criminogenic needs and how to maximize the individual's ability to profit from intervention (Andrews, Bonta, & Wormith, 2011). Finally, the aforementioned *structured professional judgment* approach formulates risk by means of a decision theory analysis of past violence and a scenario planning (Hart & Logan, 2011).

Ideally, risk formulations should express relevant risk factors, suggest probable causal pathways, speculate about future violence, and plan interventions. Some risk assessment instruments do call for the use of such formulations. For example, the LS/CMI, based on the risk-needs-responsivity approach, provides an opportunity to create an individual risk-need profile and a case management plan (Andrews et al., 2004); and HCR-20^{V3} assessments include a formulation of risk, plausible scenarios, and interventions (K. S. Douglas et al., 2013). These strategies also allow for consideration of the issues that this project has dealt with: how should individual factors like violent ideation, victimization, and health care needs be managed to reduce the risk of violent perpetration?

To put it simply: no two persons are alike, and no context in which violence occurs is identical to another. Thus, real-life assessments of violence risk have to be done on an individual level.

3.6 FUTURE DIRECTIONS

Violence risk assessments with individual risk formulations aim not only at the mere prediction of violence, but also at its prevention. Obvious as it may seem, how is this preventive ability tested empirically? A four-step procedure has been proposed: (1) validation of the selecting of risk factors, (2) demonstration of the association between risk factors and violence, (3) demonstration of the association between the clinical decisions based on the risk factors and violence, and (4) demonstration of a prevention or reduction of violence as a result of using the model in question (K. S. Douglas & Kropp, 2002). Most research appears to focus on the first two steps, especially the second. The third step may be evaluated using proxies for violence; conceivable outcome variables in a forensic psychiatric context could be use of psychological interventions, anti-aggressive medication, or restraint. At the fourth step, studies may follow the status of risk factors in response to actions taken to reduce them.

More specifically, research with an experimental clinical trial design to evaluate prevention-based violence risk assessment may be suitable (K. S. Douglas & Kropp, 2002). Patients could be randomized to one intervention group and one treatment-as-usual group. After an initial risk assessment, the intervention group could be stratified according to risk category. Interventions would then be applied on the basis of risk category and dynamic risk factors. Next, a new risk assessment would be conducted, again followed by stratification and interventions. Ultimately, the intervention group and treatment-as-usual group would be compared with respect to violence.

It is time to shift the focus away from prediction of violence among groups towards prevention of violence in the individual case. Even if predictive methods still need to be improved, a complete predictive performance is not probable because preventive measures are—and ought to be—taken in real life. Some new risk assessment instruments aim at encompassing both prediction and prevention, but empirical evaluation of the latter is still in its nascency.

3.7 CONCLUSION

Violent perpetration and violent victimization are both common among offenders with mental disorders in Sweden. Both phenomena are important to consider, especially as previous research indicates that they are associated with one another.

The risk assessment instruments COVR, LSI-R, HCR-20^{V3}, and SAPROF may be suitable to predict future violent perpetration in a Swedish forensic psychiatric context. Violent ideation alone does not appear to be a sufficient predictor; other risk factors must also be considered.

Assessment of violence risk is a prerequisite of violence prevention, but it must be supplemented with an individualized risk management plan, in which the interventions are directly derived from the risk factors.

As for violence among offenders with mental disorders, the health care services are pivotal. These services should not only assess the risk of violent perpetration and implement

preventive measures, but also provide protection from violent victimization. However, as many offenders with mental disorders report unmet health care needs, improved health care services are called for. Specialized interdisciplinary outreach teams without complicated referral procedures may be an option.

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5 APPENDIX

This appendix provides brief description of the statistical methods used in the thesis project.

ANOVA. Analysis of variance is a parametrical way of comparing means between at least three samples, where multiple *t*-tests would pose an increased risk of committing a type I error, i.e. of an incorrect rejection of a true null hypothesis. Normally distributed residuals and equal variances are assumed. The variance between samples is divided by the variance within samples to get an *F* value, more specifically

$$F = \frac{\frac{SSB}{dfB}}{\frac{SSW}{dfW}}$$

where the numerator is the sum of squares between groups divided by the degrees of freedom (number of samples - 1), and the denominator is the sum of squares within groups divided by the degrees of freedom (observations - samples). The *F* value is then compared with a critical value for an *F* distribution with these degrees of freedom, obtained from a table for the α in question (usually 0.05). ANOVA allows determination of whether one has an overall difference between groups, but to tell which specific groups differ, post hoc tests are needed.

Bonferroni correction. The Bonferroni correction is a method to adjust the α level in order to reduce the risk of type I error, which may result from multiple comparisons. To conduct a Bonferroni correction, the α level is divided by the number of hypotheses.

χ^2 test. This is a non-parametrical test used to determine whether there is a significant difference of proportions or frequencies between groups. The test statistic χ^2 is the sum of squares of the observed frequencies minus the expected frequencies divided by the expected frequencies

$$\chi^2 = \Sigma \frac{(O - E)^2}{E}$$

A χ^2 distribution table then displays the *p* value associated with the χ^2 and degrees of freedom in question. However, the sampling distribution of χ^2 is only approximately equal to the theoretical χ^2 distribution, and for small expected frequencies this approximation is inadequate.

Cohen's κ. This method is used to test the agreement between two raters measuring categorical data. It is defined as

$$\kappa = \frac{p_o - p_e}{1 - p_e}$$

where p_o is the observed percentage of agreement and p_e is the expected percentage of agreement by chance alone. When the data categories are ordinal, the degree of disagreement

may be weighted: the higher the disagreement the higher the weight. κ ranges from 1 to −1, where 1 represents a perfect agreement and -1 a perfect disagreement. A κ value of <0.20 indicates poor agreement, 0.21-0.40 fair, 0.41-0.60 moderate, 0.61-0.80 good and 0.81-1.00 very good agreement.

Conditional logistic regression. Matched data often call for certain statistical methods. The matching gives rise to dummy variables to represent each pair or set. Conducting a regular logistic regression for matched pair data tend to result in falsely overestimated squared odds ratios.

Confidence intervals. A confidence interval is a range that for repeated sampling includes an unknown population parameter with a frequency that is determined by confidence level. The confidence level is 1 - α, usually 95%. This means that out of 100 samples, 95 of them will include the parameter within the range. Not only the confidence level, but also the sample size and the distribution spread, affect this range. Confidence intervals are used to assess whether a sample differs from the population or from another sample regarding e.g. means, proportions, or odds ratios.

Fisher's exact test. For small samples, as when more than 20% of the expected frequencies are below five, Fisher's exact test supersedes the χ^2 test. This method calculates the probability of each table with the same row totals and column totals as the table in question. The probabilities of getting results as extreme as the observed are summed to get the one-sided *p* value. There are different approaches to get the two-sided p-value, the simplest is to just double the one-sided value.

Intraclass correlation. This method is used to test the agreement between raters measuring continual data. Based on ANOVA, it is in general a ratio of the between subject variance to the total variance. It consists of several models, depending on the raters. In ICC1 (SPSS: One-Way Random), each subject is rated by a different set of randomly selected raters. ICC2 (SPSS: Two-Way Random) is used when each subject is rated by each rater, and raters are randomly selected. Finally, ICC3 (SPSS: Two-way mixed) is employed when each subject is rated by each rater, and these are the only raters of interest. Agreement calculated on single measurements is denoted by ICC (1,1), ICC(2,1) and ICC(3,1), whereas ICC(1,k), ICC(2,k) and ICC(3,k) reflect the means of k raters. For the vast majority of applications, ICC(2,1) is used.

Levene's test. ANOVAs and *t*-tests assume homoscedasticity, that is equal variances across groups. Levene's test is used to assess this equality. The test is quite complicated, but it provides an *F* statistic, and if $p > 0.05$ variances are equal.

Logistic regression. In a logistic regression, a dichotomous outcome variable *y* is a function of an independent variable *x*. Usually, *y* indicates whether an event happens (1) or does not happen (0); a dummy variable may be created for this purpose. The method enables

calculation of odds ratios, showing how a one-unit change in *x* changes the odds that *y* will happen.

Mann-Whitney *U* test. This is a non-parametrical test used to determine whether there is a significant difference of location parameters, such as medians, between samples. The observations are ranked, and for each sample the ranks are summed. The test statistic *U* for each group is calculated as

$$U = R - \frac{n(n+1)}{2}$$

where *R* is the rank sum and *n* is the sample size. The smallest *U* is chosen and compared to the critical value of *U* specific to group sizes and level of significance, obtained from a table.

Odds ratio. The odds ratio *OR* measures the association between an exposure and an outcome. Odds are defined as the probability *p* that an event will take place divided by the probability that it will not take place. In an odds ratio, the numerator is the odds in the exposed group whereas the denominator is the odds in the non-exposed group:

$$OR = \frac{\frac{p_e}{1-p_e}}{\frac{p_{ne}}{1-p_{ne}}}$$

When events are rare, the odds ratio approximates the relative risk. To handle the risk of confounding, the crude odds ratio may be adjusted, i.e. by means of stratification or multivariate analyses.

Pearson product-moment correlation. The correlation coefficient *r* measures the degree of linear association between two continuous variables, *x* and *y*, that are normally distributed. The formula is

$$r = \frac{n\sum xy - \sum x \sum y}{\sqrt{n\sum x^2 - (\sum x)^2}\sqrt{n\sum y^2 - (\sum y)^2}}$$

where *n* is the number of subjects. The *t*-test may be used to assess whether the coefficient is significantly different from 0, and Fisher's *r* to *z* transformation to assess whether two coefficients are significantly different from each other.

φ coefficient. The φ coefficient is a measure of association between two binary variables. For a 2x2 table in which a, b, c, and d are the proportions occupying the four cells, the formula is

$$\Phi = \frac{ad - bc}{\sqrt{(a+b)(c+d)(a+c)(b+d)}}$$

Power calculations. The statistical power of a test consists of the probability of correctly rejecting a false H_0. It is the inverse proportion to β, the probability of making a type II error, i.e. incorrectly retaining a false H_0. Statistical power is also determined by the study design,

by α (the probability of making a type I error, i.e. incorrectly rejecting a true H_0), by the effect size (usually the difference of means of the sampling distributions of H_0 and H_a), and by the size and dispersion of the samples.

Probability value. A probability value, or *p* value, denotes the probability of obtaining a result equal to (or more extreme than) the observed result if H_0 is true.

ROC analysis. This method is used for evaluation of diagnostic tests. The true positive rate (sensitivity) of the test is plotted against the false positive rate (1 - specificity) for different cut-off points. The area under this curve (AUC) is a measure of the predictive validity of the test. A ROC curve close to the upper left corner implies an AUC close to 1, this in turn means a considerable validity. A guideline, albeit arbitrary, to interpret AUC values, is shown in Table 9.

Table 9. Interpretation of AUC values.

AUC	Interpretation
1.0	Perfect
0.90-0.99	Excellent
0.80-0.89	Good
0.70-0.79	Fair
0.51-0.69	Poor

Spearman rank-order correlation. This correlation coefficient *r* measures the degree of association between ranked, usually ordinal, variables. The formula is

$$r = 1 - \frac{6 \sum d^2}{n(n^2 - 1)}$$

where *d* is the difference between the ranks of the corresponding values of *x* and *y*, and *n* the number of subjects.

Student's *t*-test. This parametrical hypothesis test estimates whether two groups have significantly different values such as means or regression line slopes. There are several variations on this test. One-sample tests compare a sample with a population, and two-sample tests compare two samples with each other. The two-sample tests are further divided according to paired or un-paired samples, equal or unequal sample sizes, and equal or unequal variances. For an un-paired two-sample test with equal sample sizes and equal variances, the formula is

$$t = \frac{\bar{x}_1 - \bar{x}_2}{s\sqrt{\frac{1}{n}}}$$

where $\bar{x}_1$ and $\bar{x}_2$ are the sample means, s the pooled standard deviation, and n the number of subjects. The t value is then compared with a critical tabled t value for the concomitant α value, degrees of freedom, and number of tails.

Tukey's test. This test is carried out after an ANOVA, to determine which groups in a sample differ. The formula is

$$q = \frac{m_a - m_b}{\sqrt{\frac{\frac{MS_{error}}{n_a} + \frac{MS_{error}}{n_b}}{2}}}$$

where m_a and m_b are the two means, MS_{error} is the mean square of error, and n_a and n_b are the group sizes. The null hypothesis is rejected if q is larger than a critical tabled value.

Wald test. This parametrical test is used to test hypotheses. In a logistic regression, it may be used to evaluate the significance of the regression coefficients, analogous to the t-test in a linear regression. The Wald statistic, calculated as the squared coefficient divided by the squared standard error of the coefficient, is compared with a χ^2 distribution.

6 REFERENCES

Abidin, Z., Davoren, M., Naughton, L., Gibbons, O., Nulty, A., & Kennedy, H. G. (2013). Susceptibility (risk and protective) factors for in-patient violence and self-harm: Prospective study of structured professional judgement instruments START and SAPROF, DUNDRUM-3 and DUNDRUM-4 in forensic mental health services. *BMC Psychiatry, 13*, 197. doi:10.1186/1471-244X-13-197

Ægisdóttir, S., White, M. J., Spengler, P. M., Maugherman, A. S., Anderson, L. A., Cook, R. S., . . . Rush, J. D. (2006). The meta-analysis of clinical judgment project: Fifty-six years of accumulated research on clinical versus statistical prediction. *The Counseling Psychologist, 34*(3), 341-382. doi:10.1177/0011000005285875

Agnew, R., & Kaufman, J. M. (2010). *Anomie, strain and subcultural theories of crime*. Burlington, VT: Ashgate.

Al Odhayani, A., Watson, W. J., & Watson, L. (2013). Behavioural consequences of child abuse. *Canadian Family Physician, 59*(8), 831-836.

Aldridge, A. (2013). *Religion in the contemporary world: A sociological introduction* (3rd ed.). Malden, MA: Polity.

Allely, C. S., Wilson, P., Minnis, H., Thompson, L., Yaksic, E., & Gillberg, C. (2017). Violence is rare in autism: When it does occur, is it sometimes extreme? *Journal of Psychology, 151*(1), 49-68. doi:10.1080/00223980.2016.1175998

Anderson, C. A., & Bushman, B. J. (2002). Human aggression. *Annual Review of Psychology, 53*, 27-51. doi:10.1146/annurev.psych.53.100901.135231

Anderson, N. H. (2001). *Empirical direction in design and analysis*. Mahwah, NJ: L. Erlbaum.

Andrade, J. T. (2009). *Handbook of violence risk assessment and treatment: New approaches for mental health professionals*. New York, NY: Springer.

Andrews, D. A., & Bonta, J. L. (1995). *LSI-R™. Level of Service Inventory-Revised.* Toronto, ON: Multi-Health Systems.

Andrews, D. A. (1982). *The Level of Supervisory Inventory (LSI): 1. The first follow-up.* Toronto, ON: Ontario Ministry of Correctional Services, Planning and Support Services Division, Planning and Research Branch.

Andrews, D. A., & Bonta, J. L. (1995). *Level of Service Inventory-Revised: Screening Version (LSI-R:SV): User's manual*. Toronto, ON: Multi-Health Systems.

Andrews, D. A., Bonta, J. L., & Wormith, S. J. (2006). The recent past and near future of risk and/or need assessment. *Crime & Delinquency, 52*(1), 7-27. doi:10.1177/0011128705281756

Andrews, D. A., Bonta, J. L., & Wormith, S. J. (2010). The Level of Service (LS) assessment of adults and older adolescents. In Randy K Otto & Kevin S Douglas (Eds.), *Handbook of violence risk assessment*. New York, NY: Routledge.

Andrews, D. A., Bonta, J. L., & Wormith, S. J. (1995). *Level of Service Inventory-Ontario Revision (LSI-OR): Interview and scoring guide*. Toronto, ON: Ontario Ministry of the Solicitor General and Correctional Services.

Andrews, D. A., Bonta, J. L., & Wormith, S. J. (2004). *The Level of Service/Case Management Inventory (LS/CMI): User's manual*. Toronto, ON: Multi-Health Systems.

Andrews, D. A., Bonta, J. L., & Wormith, S. J. (2011). The Risk-Need-Responsivity model. *Criminal Justice and Behavior, 38*(7), 735-755. doi:10.1177/0093854811406356

Andrews, D. A., Robinson, D., & Hoge, R. D. (1984). *Manual for the Youth Level of Service Inventory*. Ottawa, ON: Department of Psychology, Carleton University.

Archer, J. (1991). The influence of testosterone on human aggression. *British Journal of Psychology, 82 (1)*, 1-28.

Archer, J., Graham-Kevan, N., & Davies, M. (2005). Testosterone and aggression: A reanalysis of Book, Strarzyk, and Quinsey's (2001) study. *Aggression and Violent Behavior, 10*(2), 241-261. doi:10.1016/j.avb.2004.01.001

Aristotle, Bartlett, R. C., & Collins, S. D. (2011). *Aristotle's Nicomachean ethics*. Chicago, IL: University of Chicago Press.

Arnold, T. K. (2007). *Dynamic changes in the Level of Service Inventory-Revised (LSI-R) and the effects on prediction accuracy.* (Master of Science), St. Cloud State University, St. Cloud, Minnesota.

Asghar, K., De Souza, E. B., & National Institute on Drug Abuse. (1989). *Pharmacology and toxicology of amphetamine and related designer drugs*. Washington, DC: U.S. Dept. of Health and Human Services, Public Health Service, Alcohol, Drug Abuse, and Mental Health Administration

Audet, M. C., Goulet, S., & Doré, F. Y. (2009). Impaired social motivation and increased aggression in rats subchronically exposed to phencyclidine. *Physiology and Behavior, 96*(3), 394-398. doi:10.1016/j.physbeh.2008.11.002

Austin, P. C. (2011a). An introduction to propensity score methods for reducing the effects of confounding in observational studies. *Multivariate Behavioral Research, 46*(3), 399-424. doi:10.1080/00273171.2011.568786

Austin, P. C. (2011b). A tutorial and case study in propensity score analysis: An application to estimating the effect of in-hospital smoking cessation counseling on mortality. *Multivariate Behavioral Research, 46*(1), 119-151. doi:10.1080/00273171.2011.540480

Bandura, A. (1973). Social learning theory of aggression. In John F. Knutson (Ed.), *The control of aggression: Implications from basic research* (pp. 310 p.). Chicago, IL: Aldine Pub. Co.

Bandura, A. (1977). *Social learning theory*. Englewood Cliffs, NJ: Prentice Hall.

Bandura, A., Ross, D., & Ross, S. A. (1961). Transmission of aggression through imitation of aggressive models. *Journal of Abnormal and Social Psychol, 63*(3), 575-582. doi:10.1037/h0045925

Bandura, A., Ross, D., & Ross, S. A. (1963). Imitation of film-mediated aggressive models. *The Journal of Abnormal and Social Psychology, 66*(1), 3-11. doi:10.1037/h0048687

Baron, R. A., & Richardson, D. S. (1994). *Human aggression* (2nd ed.). New York, NY: Plenum Press.

Barton-Bellessa, S. M. (2012). *Encyclopedia of community corrections*. Thousand Oaks, CA: SAGE Publications.

Beauchamp, T. L., & Childress, J. F. (1979). *Principles of biomedical ethics*. New York, NY: Oxford University Press.

Beaver, K. M., Vaughn, M. G., Delisi, M., & Wright, J. P. (2008). Anabolic-androgenic steroid use and involvement in violent behavior in a nationally representative sample of young adult males in the United States. *American Journal of Public Health, 98*(12), 2185-2187. doi:10.2105/AJPH.2008.137018

Belfrage, H., Strand, S., Storey, J. E., Gibas, A. L., Kropp, P. R., & Hart, S. D. (2012). Assessment and management of risk for intimate partner violence by police officers using the Spousal Assault Risk Assessment guide. *Law and Human Behavior, 36*(1), 60-67. doi:10.1007/s10979-011-9278-0

Benarroch, E. E. (2012). Periaqueductal gray: An interface for behavioral control. *Neurology, 78*(3), 210-217. doi:10.1212/WNL.0b013e31823fcdee

Berkowitz, L. (1989). Frustration-aggression hypothesis: Examination and reformulation. *Psychological Bulletin, 106*(1), 59-73.

Berkowitz, L. (1990). On the formation and regulation of anger and aggression. A cognitive-neoassociationistic analysis. *American Psychologist, 45*(4), 494-503.

Bevilacqua, L., Doly, S., Kaprio, J., Yuan, Q., Tikkanen, R., Paunio, T., . . . Goldman, D. (2010). A population-specific HTR2B stop codon predisposes to severe impulsivity. *Nature, 468*(7327), 1061-1066. doi:10.1038/nature09629

Bhakta, S. G., Zhang, J. P., & Malhotra, A. K. (2012). The COMT Met158 allele and violence in schizophrenia: A meta-analysis. *Schizophrenia Research, 140*(1-3), 192-197. doi:10.1016/j.schres.2012.06.026

Bjerrum Moeller, S., Gondan, M., & Novaco, R. W. (2017). Violent images, anger and physical aggression among male forensic inpatients. *Personality and Individual Differences, 105*, 268-274. doi:10.1016/j.paid.2016.10.009

Bjørkly, S. (2013). A systematic review of the relationship between impulsivity and violence in persons with psychosis: Evidence or spin cycle? *Aggression and Violent Behavior, 18*(6). doi:10.1016/j.avb.2013.08.001

Bleuler, E. (1896). *Der geborene Verbrecher. Eine kritische Studie*. Munich, Germany: Verlag von J. F. Lehmann

Blitz, C. L., Wolff, N., & Shi, J. (2008). Physical victimization in prison: The role of mental illness. *International Journal of Law and Psychiatry, 31*(5), 385-393. doi:10.1016/j.ijlp.2008.08.005

Blomquist, C. (1978). Declaration of Hawaii. *Journal of Medical Ethics, 4*(2), 71-73.

Blumer, H. (1969). *Symbolic interactionism: Perspective and method*. Englewood Cliffs, NJ: Prentice-Hall.

Bodhi, B., & Nanamoli, B.. (2015). *The middle length discourses of the Buddha: A translation of the Majjhima Nikaya*. Somerville: MA: Wisdom Publications.

Boer, D. P., Hart, S. D., Kropp, P. R., & Webster, C. D. (1997). *Manual for the Sexual Violence Risk-20*. Burnaby, BC: Simon Fraser University.

Bohm, R. M., & Vogel, B. (2015). *A primer on crime and delinquency theory* (4th ed.). Durham, NC: Carolina Academic Press.

Bohman, M. (1996). Predisposition to criminality: Swedish adoption studies in retrospect. *Ciba Foundation Symposium, 194*, 99-109; discussion 109-114.

Bohnen, I., Bakala, A., Thakker, Y., & Wijeratne, A. (2013). Management of violence in people with intellectual disability. In Masum Khwaja & Dominic Beer (Eds.), *Prevention and management of violence: Guidance for mental healthcare professionals*. London, UK: RCPsych Publications.

Bonta, J. L., Blais, J., & Wilson, H. A. (2014). A theoretically informed meta-analysis of the risk for general and violent recidivism for mentally disordered offenders. *Aggression and Violent Behavior, 19*(3), 278-287. doi:10.1016/j.avb.2014.04.014

Book, A. S., Starzyk, K. B., & Quinsey, V. L. (2001). The relationship between testosterone and aggression: A meta-analysis. *Aggression and Violent Behavior, 6*(6), 579-599. doi:10.1016/S1359-1789(00)00032-X

Bowers, D., House, A., & Owens, D. (2011). *Getting started in health research*. Hoboken, NJ: Wiley-Blackwell/BMJ Books.

Bowers, L., Stewart, D., Papadopoulos, C., Dack, C., Ross, J., Khanom, H., & Jeffrey, D. (2011). *Inpatient violence and aggression: A literature review*. Retrieved from King's College website: http://www.kcl.ac.uk/ioppn/depts/hspr/research/ciemh/mhn/projects/litreview/LitRevAgg.pdf

Brennan, P. A., Mednick, S. A., & Jacobsen, B. (1996). Assessing the role of genetics in crime using adoption cohorts. *Ciba Foundation Symposium, 194*, 115-123; discussion 123-128.

Broderick, C., Azizian, A., Kornbluh, R., & Warburton, K. (2015). Prevalence of physical violence in a forensic psychiatric hospital system during 2011-2013: Patient assaults, staff assaults, and repeatedly violent patients. *CNS Spectrums, 20*(3), 319-330. doi:10.1017/S1092852915000188

Brunner, H. G., Nelen, M., Breakefield, X. O., Ropers, H. H., & van Oost, B. A. (1993). Abnormal behavior associated with a point mutation in the structural gene for monoamine oxidase A. *Science, 262*(5133), 578-580.

Buchanan, A., & Grounds, A. (2011). Forensic psychiatry and public protection. *British Journal of Psychiatry, 198*(6), 420-423. doi:10.1192/bjp.bp.111.095471

Bufkin, J. L., & Luttrell, V. R. (2005). Neuroimaging studies of aggressive and violent behavior: Current findings and implications for criminology and criminal justice. *Trauma Violence Abuse, 6*(2), 176-191. doi:10.1177/1524838005275089

Bunge, M. (1985). *Philosophy of science and technology*. Boston, MA: Kluwer Academic.

Bushman, B. J., & Anderson, C. A. (2001). Is it time to pull the plug on the hostile versus instrumental aggression dichotomy? *Psychological Review, 108*(1), 273-279.

Butovskaya, M. L., Vasilyev, V. A., Lazebny, O. E., Suchodolskaya, E. M., Shibalev, D. V., Kulikov, A. M., . . . Ryskov, A. P. (2013). Aggression and polymorphisms in AR, DAT1, DRD2, and COMT genes in Datoga pastoralists of Tanzania. *Scientific Reports, 3*, 3148. doi:10.1038/srep03148

Cannon, M., Huttunen, M. O., Tanskanen, A. J., Arseneault, L., Jones, P. B., & Murray, R. M. (2002). Perinatal and childhood risk factors for later criminality and violence in schizophrenia. Longitudinal, population-based study. *British Journal of Psychiatry, 180*, 496-501.

Carey, G. (1994). Genetics and violence. In Albert J Reiss, Klaus A Miczek, & Jeffrey A Roth (Eds.), *Understanding and preventing violence* (Vol. 2). Washington, DC: National Academies Press.

Cartwright, J. (2016). *Evolution and human behaviour: Darwinian perspectives on the human condition*. London, UK: Palgrave.

Caspi, A., McClay, J., Moffitt, T. E., Mill, J., Martin, J., Craig, I. W., . . . Poulton, R. (2002). Role of genotype in the cycle of violence in maltreated children. *Science, 297*(5582), 851-854. doi:10.1126/science.1072290

Cautin, R. L. editor, & Lilienfeld, S. O. editor. (2015). *The encyclopedia of clinical psychology*. New York, NY: Wiley-Blackwell.

Choe, J. Y., Teplin, L. A., & Abram, K. M. (2008). Perpetration of violence, violent victimization, and severe mental illness: Balancing public health concerns. *Psychiatric Services, 59*(2), 153-164. doi:10.1176/appi.ps.59.2.153

Christiansen, E. J., & Evans, W. P. (2005). Adolescent victimization: Testing models of resiliency by gender. *The Journal of Early Adolescence, 25*(3), 298-316. doi:10.1177/0272431605276931

Cicero, M. T., & Edmonds, C. R. (1850). *Cicero's three books of offices, or moral duties*. London, UK: H. G. Bohn.

Coccaro, E. F., Lee, R., & Coussons-Read, M. (2014). Elevated plasma inflammatory markers in individuals with intermittent explosive disorder and correlation with aggression in humans. *JAMA Psychiatry, 71*(2), 158-165. doi:10.1001/jamapsychiatry.2013.3297

Coccaro, E. F., Lee, R., & Coussons-Read, M. (2015). Cerebrospinal fluid inflammatory cytokines and aggression in personality disordered subjects. *International Journal of Neuropsychopharmacology, 18*(7), pyv001. doi:10.1093/ijnp/pyv001

Coccaro, E. F., Lee, R., Liu, T., & Mathé, A. A. (2012). Cerebrospinal fluid neuropeptide Y-like immunoreactivity correlates with impulsive aggression in human subjects. *Biological Psychiatry, 72*(12), 997-1003. doi:10.1016/j.biopsych.2012.07.029

Coccaro, E. F., Lee, R., Owens, M. J., Kinkead, B., & Nemeroff, C. B. (2012). Cerebrospinal fluid substance P-like immunoreactivity correlates with aggression in personality disordered subjects. *Biological Psychiatry, 72*(3), 238-243. doi:10.1016/j.biopsych.2012.02.023

Cohen, J. (1992). A power primer. *Psychological Bulletin, 112*(1), 155-159.

Cohen, J. (1988). *Statistical power analysis for the behavioral sciences* (2nd ed.). Hillsdale, NJ: L. Erlbaum Associates.

Cohen, L. E., & Felson, M. (1979). Social change and crime rate trends: A routine activity approach. *American Sociological Review, 44*, 588-608.

Coid, J., Yang, M., Ullrich, S., Zhang, T., Sizmur, S., Roberts, C., . . . Rogers, R. D. (2009). Gender differences in structured risk assessment: Comparing the accuracy of five

instruments. *Journal of Consulting and Clinical Psychology, 77*(2), 337-348. doi:10.1037/a0015155

Cornish, D. B., & Clarke, R. V. G. (2014). *The reasoning criminal: Rational choice perspectives on offending*. New York, NY: Transaction Publishers.

Corsini, R. J., & Wedding, D. (2010). *Current psychotherapies* (9th ed.). Belmont, CA: Brooks/Cole, Cengage Learning.

Craig, I. W. (2007). The importance of stress and genetic variation in human aggression. *Bioessays, 29*(3), 227-236. doi:10.1002/bies.20538

Cusack, K. J., Frueh, B. C., & Brady, K. T. (2004). Trauma history screening in a community mental health center. *Psychiatric Services, 55*(2), 157-162.

Dack, C., Ross, J., Papadopoulos, C., Stewart, D., & Bowers, L. (2013). A review and meta-analysis of the patient factors associated with psychiatric in-patient aggression. *Acta Psychiatrica Scandinavica, 127*(4), 255-268. doi:10.1111/acps.12053

Daff, E. S., Gilbert, F., & Daffern, M. (2015). The relationship between anger and aggressive script rehearsal in an offender population. *Psychiatry, Psychology and Law, 22*(5), 731-739. doi:10.1080/13218719.2014.986837

Daffern, M., Jones, L., & Shine, J. (2010). *Offence paralleling behaviour: A case formulation approach to offender assessment and intervention*. Hoboken, NJ: Wiley.

Daigle, L. E., Beaver, K. M., & Turner, M. G. (2010). Resiliency against victimization: Results from the National Longitudinal Study of Adolescent Health. *Journal of Criminal Justice, 38*(3), 329-337. doi:10.1016/j.jcrimjus.2010.03.007

Daigle, L. E. (2012). *Victimology: A text/reader*. Thousand Oaks, CA: SAGE Publications.

Darwin, C. (1859). *On the origin of species by means of natural selection, or the preservation of favoured races in the struggle for life*. London, UK: John Murray.

Das, S., Deuri, S. K., Sarmah, A., Pathak, K., Baruah, A., Sengupta, S., . . . Hazarika, J. (2016). Aggression as an independent entity even in psychosis: The role of inflammatory cytokines. *Journal of Neuroimmunology, 292*, 45-51. doi:10.1016/j.jneuroim.2016.01.012

Dawe, S., Davis, P., Lapworth, K., & McKetin, R. (2009). Mechanisms underlying aggressive and hostile behavior in amphetamine users. *Current Opinion in Psychiatry, 22*(3), 269-273. doi:10.1097/YCO.0b013e32832a1dd4

de Graaf, M. A., Jager, K. J., Zoccali, C., & Dekker, F. W. (2011). Matching, an appealing method to avoid confounding? *Nephron: Clinical Practice, 118*(4), 315-318. doi:10.1159/000323136

de Vaan, M. A. C. (2008). *Etymological dictionary of Latin and the other Italic languages*. Boston, MA: Brill.

de Vogel, V., de Ruiter, C., Bouman, Y., & de Vries Robbé, M. (2009). *SAPROF. Guidelines for the assessment of protective factors for violence risk. English version.* Utrecht, the Netherlands: Forum Educatief.

de Vogel, V., de Ruiter, C., Bouman, Y., & de Vries Robbé, M. (2007). *SAPROF. Richtlijnen voor het beoordelen van beschermende factoren voor gewelddadig gedrag. Versie 1.* Utrecht, the Netherlands: Forum Educatief.

de Vogel, V., de Ruyter, C., & Bouman, Y. (2004). *Scoringshandleiding SAPROF. Structured Assessment of Violence Risk. Research Versie.* Utrecht, the Netherlands: Van der Hoevek Kliniek, Trimbos Instituut, Pompestichting.

de Vries Robbé, M. (2014). *Protective factors. Validation of the Structured assessment of protective factors for violence risk in forensic psychiatry.* (Doctoral dissertation), Radboud University, Nijmegen, Netherlands.

de Vries Robbé, M., de Spa, E., & de Vogel, V. (2007). *Protective factors for violence risk. Clinical experiences and first results with a new instrument for risk prevention, the Structured Assessment of Protective Factors for Violence Risk*. Paper presented at the Fifth Congress on Violence in Clinical Psychiatry, Amsterdam, the Netherlands.

de Vries Robbé, M., & de Vogel, V. (2014). *SAPROF Interview Self-Appraisal*. Utrecht, the Netherlands: Van der Hoeven Kliniek.

de Vries Robbé, M., Geers, M. C. K., Stapel, M., Hilterman, E. L. B., & de Vogel, V. (2015). *SAPROF Youth Version: Guidelines for the assessment of protective factors for violence in juveniles*. Utrecht, the Netherlands: De Forensische Zorgspecialisten.

de Vries Robbé, M., & Hilterman, E. L. B. (2015). *SAPROF Youth Version Interview Self-Appraisal*. Utrecht, the Netherlands: Van der Hoeven Kliniek.

Desmarais, S. L., Van Dorn, R. A., Johnson, K. L., Grimm, K. J., Douglas, K. S., & Swartz, M. S. (2014). Community violence perpetration and victimization among adults with mental illnesses. *American Journal of Public Health, 104*(12), 2342-2349. doi:10.2105/AJPH.2013.301680

Dharmawardene, V., & Menkes, D. B. (2017). Violence and self-harm in severe mental illness: Inpatient study of associations with ethnicity, cannabis and alcohol. *Australasian Psychiatry, 25*(1), 28-31. doi:10.1177/1039856216671650

Diamond, B. L., & Quen, J. M. (1994). *The psychiatrist in the courtroom: Selected papers of Bernard L. Diamond*. Hillsdale, NJ: Analytic Press.

DiGiuseppe, R., & Tafrate, R. C. (2007). *Understanding anger disorders*. New York, NY: Oxford University Press.

Dodge, K. A., Bates, J. E., & Pettit, G. S. (1990). Mechanisms in the cycle of violence. *Science, 250*(4988), 1678-1683.

Dollard, J., Doob, L. W., Miller, N. E., Mowrer, O. H., Sears, R. R., & Yale university Institute of human relations. (1939). *Frustration and aggression*. New Haven, CT: Yale university press

Douglas, K. S. (2014). Introduction to the special issue of the HCR-20 Version 3. *International Journal of Forensic Mental Health, 13*(2), 91-92. doi:10.1080/14999013.2014.913746

Douglas, K. S., Guy, L. S., & Hart, S. D. (2009). Psychosis as a risk factor for violence to others: A meta-analysis. *Psychological Bulletin, 135*(5), 679-706. doi:10.1037/a0016311

Douglas, K. S., Hart, S. D., Webster, C. D., & Belfrage, H. (2013). *HCR-20^{V3}. Assessing risk for violence. Version 3.* Vancouver, BC: Simon Fraser University.

Douglas, K. S., Hart, S. D., Webster, C. D., Belfrage, H., Guy, L. S., & Wilson, C. M. (2014). Historical-Clinical-Risk Management-20, version 3 (HCR-20V3): Development and

overview. *International Journal of Forensic Mental Health, 13*(2), 93-108. doi:10.1080/14999013.2014.906519

Douglas, K. S., & Kropp, P. R. (2002). A prevention-based paradigm for violence risk assessment: Clinical and research applications. *Criminal Justice and Behavior, 29*(5), 617-658. doi:10.1177/009385402236735

Douglas, K. S., & Skeem, J. L. (2005). Violence risk assessment: Getting specific about being dynamic. *Psychology, Public Policy, and Law, 11*(3), 347-383. doi:10.1037/1076-8971.11.3.347

Douglas, K. S., Webster, C. D., & Wintrup, A. M. (1996). *The HCR-20 risk assessment scheme: Psychometric properties in two samples*. Paper presented at the American Psychological Association Annual Conference, Toronto, Ontario.

Douglas, T., Pugh, J., Singh, I., Savulescu, J., & Fazel, S. (2017). Risk assessment tools in criminal justice and forensic psychiatry: The need for better data. *European Psychiatry, 42*, 134-137. doi:10.1016/j.eurpsy.2016.12.009

Doyle, M., Shaw, J., Carter, S., & Dolan, M. (2010). Investigating the validity of the Classification of Violence Risk in a UK sample. *International Journal of Forensic Mental Health, 9*(4), 316-323. doi:10.1080/14999013.2010.527428

Duke, A. A., Bègue, L., Bell, R., & Eisenlohr-Moul, T. (2013). Revisiting the serotonin-aggression relation in humans: A meta-analysis. *Psychological Bulletin, 139*(5), 1148-1172. doi:10.1037/a0031544

Dutton, M. A., & Greene, R. (2010). Resilience and crime victimization. *Journal of Traumatic Stress, 23*(2), 215-222. doi:10.1002/jts.20510

Egan, S. K., & Perry, D. G. (1998). Does low self-regard invite victimization? *Developmental Psychology, 34*(2), 299-309.

Eglin, P., & Hester, S. (2017). *A sociology of crime* (2nd ed.). New York, NY: Routledge.

Eitle, D. (2010). General strain theory, persistence, and desistance among young adult males. *Journal of Criminal Justice, 38*(6), 1113-1121. doi:10.1016/j.jcrimjus.2010.08.003

Elbogen, E. B., Van Dorn, R. A., Swanson, J. W., Swartz, M. S., & Monahan, J. (2006). Treatment engagement and violence risk in mental disorders. *British Journal of Psychiatry, 189*, 354-360. doi:10.1192/bjp.bp.105.017913

ElSohly, M. A. (2007). *Marijuana and the cannabinoids*. Totowa, NJ: Humana Press.

Emerson, E., Kiernan, C., Alborz, A., Reeves, D., Mason, H., Swarbrick, R., . . . Hatton, C. (2001). The prevalence of challenging behaviors: A total population study. *Research in Developmental Disabilities, 22*(1), 77-93.

Falkner, A. L., Grosenick, L., Davidson, T. J., Deisseroth, K., & Lin, D. (2016). Hypothalamic control of male aggression-seeking behavior. *Nature Neuroscience, 19*(4), 596-604. doi:10.1038/nn.4264

Falkner, A. L., & Lin, D. (2014). Recent advances in understanding the role of the hypothalamic circuit during aggression. *Frontiers in Systems Neuroscience, 8*, 168. doi:10.3389/fnsys.2014.00168

Fan, J., Upadhye, S., & Worster, A. (2006). Understanding receiver operating characteristic (ROC) curves. *Canadian Journal of Emergency Medicine, 8*(1), 19-20.

Farrington, D. P., & Murray, J. (2014). *Labeling theory: Empirical tests*. New Brunswick, NJ: Transaction Publishers.

Fazel, S., & Grann, M. (2006). The population impact of severe mental illness on violent crime. *The American Journal of Psychiatry, 163*(8), 1397-1403. doi:10.1176/appi.ajp.163.8.1397

Fazel, S., Gulati, G., Linsell, L., Geddes, J. R., & Grann, M. (2009). Schizophrenia and violence: Systematic review and meta-analysis. *PLoS Medicine, 6*(8), e1000120. doi:10.1371/journal.pmed.1000120

Fazel, S., Lichtenstein, P., Frisell, T., Grann, M., Goodwin, G., & Långström, N. (2010). Bipolar disorder and violent crime: Time at risk reanalysis. *Archives of General Psychiatry, 67*(12), 1325-1326. doi:10.1001/archgenpsychiatry.2010.171

Fazel, S., Lichtenstein, P., Grann, M., Goodwin, G. M., & Långström, N. (2010). Bipolar disorder and violent crime: New evidence from population-based longitudinal studies and systematic review. *Archives of General Psychiatry, 67*(9), 931-938. doi:10.1001/archgenpsychiatry.2010.97

Fazel, S., Lichtenstein, P., Grann, M., & Långström, N. (2011). Risk of violent crime in individuals with epilepsy and traumatic brain injury: A 35-year Swedish population study. *PLoS Medicine, 8*(12), e1001150. doi:10.1371/journal.pmed.1001150

Fazel, S., Singh, J. P., Doll, H., & Grann, M. (2012). Use of risk assessment instruments to predict violence and antisocial behaviour in 73 samples involving 24 827 people: Systematic review and meta-analysis. *BMJ, 345*, e4692. doi:10.1136/bmj.e4692.

Fazel, S., Wolf, A., Chang, Z., Larsson, H., Goodwin, G. M., & Lichtenstein, P. (2015). Depression and violence: A Swedish population study. *Lancet Psychiatry, 2*(3), 224-232. doi:10.1016/S2215-0366(14)00128-X

Fazel, S., & Yu, R. (2011). Psychotic disorders and repeat offending: Systematic review and meta-analysis. *Schizophrenia Bulletin, 37*(4), 800-810. doi:10.1093/schbul/sbp135

Fazel, S., & Bjørkly, S. (2016). Methodological considerations in risk assessment research. In Jay P Sing, Stål Bjørkly, & Seena Fazel (Eds.), *International perspectives on violence risk assessment*. New York, NY: Oxford University Press.

Ferguson, A. M., Ogloff, J. R. P., & Thomson, L. (2009). Predicting recidivism by mentally disordered offenders using the LSI-R:SV. *Criminal Justice and Behavior, 31*(1), 5-20. doi:10.1177/0093854808326525

Fink, A. (1995). *The survey kit*. Thousand Oaks, CA: SAGE Publications.

Fleury, M. J., Grenier, G., Bamvita, J. M., Perreault, M., & Caron, J. (2012). Determinants associated with the utilization of primary and specialized mental health services. *Psychiatric Quarterly, 83*(1), 41-51. doi:10.1007/s11126-011-9181-3

Florkowski, C. M. (2008). Sensitivity, specificity, receiver-operating characteristic (ROC) curves and likelihood ratios: Communicating the performance of diagnostic tests. *Clinical Biochemist, 29 Suppl 1*, S83-87.

Flynn, J. (2013). *Exploring the instrumental and reactive violence dichotomy in the offences of violent psychopaths.* (Master of science), Victoria University, Wellington, New Zealand.

Fodor, A., Barsvari, B., Aliczki, M., Balogh, Z., Zelena, D., Goldberg, S. R., & Haller, J. (2014). The effects of vasopressin deficiency on aggression and impulsiveness in

male and female rats. *Psychoneuroendocrinology, 47*, 141-150. doi:10.1016/j.psyneuen.2014.05.010

Fountoulakis, K. N., Leucht, S., & Kaprinis, G. S. (2008). Personality disorders and violence. *Current Opinion in Psychiatry, 21*(1), 84-92. doi:10.1097/YCO.0b013e3282f31137

Franzen, M. D. (2000). *Reliability and validity in neuropsychological assessment* (2nd ed.). New York, NY: Kluwer Academic/Plenum Publishers.

Freud, S., Freud, A., Strachey, A., Strachey, J., & Tyson, A. (1961). *The ego and the id, and other works; translated under the general editorship of James Strachey in collaboration with Anna Freud, assisted by Alix Strachey and Alan Tyson*. London, UK: Hogarth Press.

Frisell, T., Lichtenstein, P., & Långström, N. (2011). Violent crime runs in families: A total population study of 12.5 million individuals. *Psychological Medicine, 41*(1), 97-105. doi:10.1017/S0033291710000462

Funahashi, S. (2017). Working Memory in the Prefrontal Cortex. *Brain Sciences, 7*(5). doi:10.3390/brainsci7050049

Funahashi, S., & Andreau, J. M. (2013). Prefrontal cortex and neural mechanisms of executive function. *Journal of Physiology, Paris, 107*(6), 471-482. doi:10.1016/j.jphysparis.2013.05.001

Furukawa, T. A. (2015). The relationship between depression and violent crime. *Lancet Psychiatry, 2*(3), 193-194. doi:10.1016/S2215-0366(15)00046-2

Gendreau, P., Goggin, C., & Smith, P. (2002). Is the PCL-R really the "unparalleled" measure of offender risk? A lesson in knowledge cumulation. *Criminal Justice and Behavior, 29*(4), 397-426. doi:10.1177/0093854802029004004

Gillon, R. (1994). Medical ethics: Four principles plus attention to scope. *BMJ, 309*(6948), 184-188.

Glas, A. S., Lijmer, J. G., Prins, M. H., Bonsel, G. J., & Bossuyt, P. M. (2003). The diagnostic odds ratio: A single indicator of test performance. *Journal of Clinical Epidemiology, 56*(11), 1129-1135.

Goldstein, A. P. (1981). *In response to aggression : Methods of control and prosocial alternatives*. New York, NY: Pergamon Press.

Gómez, J. M., Verdú, M., González-Megías, A., & Méndez, M. (2016). The phylogenetic roots of human lethal violence. *Nature, 538*(7624), 233-237. doi:10.1038/nature19758

Government bill 1990/91:58. (1990). *Regeringens proposition om psykiatrisk tvångsvård*

Grant, C. (2002). *Theories of crime and punishment*. Harlow, UK: Longman.

Greenberg, G., & Haraway, M. M. (1998). *Comparative psychology: A handbook*. New York, NY: Garland Pub.

Grisso, T., Davis, J., Vesselinov, R., Appelbaum, P. S., & Monahan, J. (2000). Violent thoughts and violent behavior following hospitalization for mental disorder. *Journal of Consulting and Clinical Psychology, 68*(3), 388-398.

Guidotti, T. L. (2012). Phineas Gage and his frontal lobe-the "American Crowbar Case". *Archives of Environmental & Occupational Health, 67*(4), 249-250. doi:10.1080/19338244.2012.722469

Guo, G., Roettger, M. E., & Shih, J. C. (2007). Contributions of the DAT1 and DRD2 genes to serious and violent delinquency among adolescents and young adults. *Human Genetics, 121*(1), 125-136. doi:10.1007/s00439-006-0244-8

Haberstick, B. C., Lessem, J. M., Hewitt, J. K., Smolen, A., Hopfer, C. J., Halpern, C. T., . . . Mullan Harris, K. (2014). MAOA genotype, childhood maltreatment, and their interaction in the etiology of adult antisocial behaviors. *Biological Psychiatry, 75*(1), 25-30. doi:10.1016/j.biopsych.2013.03.028

Haden, S. C., & Scarpa, A. (2007). The noradrenergic system and its involvement in aggressive behaviors. *Aggression and Violent Behavior, 12*(1), 1-15. doi:10.1016/j.avb.2006.01.012

Haglund, A., Lindh, Å. U., Lysell, H., Renberg, E. S., Jokinen, J., Waern, M., & Runeson, B. (2016). Interpersonal violence and the prediction of short-term risk of repeat suicide attempt. *Scientific Reports, 6*, 36892. doi:10.1038/srep36892

Haller, J. (2014). *Neurobiological bases of abnormal aggression and violent behaviour*. Vienna, Austria: Springer.

Hare, R. D. (1991). *The Hare psychopathy checklist-revised*. North Tonawanda, NY: Multi-Health Systems, Inc.

Hare, R. D. (1999). Psychopathy as a risk factor for violence. *Psychiatric Quarterly, 70*(3), 181-197.

Hare, R. D. (2006). Psychopathy: A clinical and forensic overview. *Psychiatric Clinics of North America, 29*(3), 709-724. doi:10.1016/j.psc.2006.04.007

Harper, R. F. (1904). *The Code of Hammurabi, King of Babylon: About 2250 B. C.: Autographed text, transliteration, translation, glossary index of subjects, lists of proper names, signs, numuerals*. Chicago, IL: The University of Chicago Press, Callaghan & Co.

Harrendorf, S., Heiskanen, M., & Malby, S. (2010). *International statistics on crime and justice*. Helsinki, Finland: European Institute for Crime Prevention and Control in affiliation with United Nations Office on Drugs and Crime.

Harris, G. T., Rice, M. E., & Camilleri, J. A. (2004). Applying a forensic actuarial assessment (the Violence Risk Appraisal Guide) to nonforensic patients. *Journal of Interpersonal Violence, 19*(9), 1063-1074. doi:10.1177/0886260504268004

Harris, G. T., Rice, M. E., & Quinsey, V. L. (1993). Violent recidivism of mentally disordered offenders: The development of a statistical prediction instrument. *Criminal Justice and Behavior, 20*(4), 315-335.

Hart, S. D., & Logan, C. (2011). Formulation of violence risk using evidence-based assessments: The structured professional judgment approach. In Peter Sturmey & Mary McMurran (Eds.), *Forensic case formulation*. Chichester, UK: Wiley-Blackwell.

Hasan, A. (2012). *Sunan Abu Dawud: English translation with explanatory notes* (11 ed. Vol. 3). New Dehli, India: Kitab Bhavan.

Hastings, M. E., Krishnan, S., Tangney, J. P., & Stuewig, J. (2011). Predictive and incremental validity of the Violence Risk Appraisal Guide scores with male and female jail inmates. *Psychological Assessment, 23*(1), 174-183. doi:10.1037/a0021290

Helmchen, H., & Okasha, A. (2000). From the Hawaii Declaration to the Declaration of Madrid. *Acta Psychiatrica Scandinavica. Supplementum, 399*, 20-23.

Heron, M. (2016). Deaths: Leading causes for 2014. *National Vital Statistics Reports, 65*(5), 1-95.

Hobbes, T. (1996). Of Civill Lawes. In Richard Tuck (Ed.), *Leviathan* (Rev. ed.). Cambridge, UK: Cambridge University Press.

Hodgins, S., Kratzer, L., & McNeil, T. F. (2001). Obstetric complications, parenting, and risk of criminal behavior. *Archives of General Psychiatry, 58*(8), 746-752.

Hodgins, S., Kratzer, L., & McNeil, T. F. (2002). Obstetrical complications, parenting practices and risk of criminal behaviour among persons who develop major mental disorders. *Acta Psychiatrica Scandinavica, 105*(3), 179-188.

Hogan, N. R., & Olver, M. E. (2016). Assessing risk for aggression in forensic psychiatric inpatients: An examination of five measures. *Law and Human Behavior, 40*(3), 233-243. doi:10.1037/lhb0000179

Howard, R. C. (2017). Psychopathy, impulsiveness and violence: How are they linked? *Journal of Behavior, 2*(1), 1004.

Howells, K., Day, A., Williamson, P., Bubner, S., Jauncey, S., Parker, A., & Heseltine, K. (2005). Brief anger management programs with offenders: Outcomes and predictors of change. *The Journal of Forensic Psychiatry & Psychology, 16*(2), 296-311. doi:10.1080/14789940500096099

Huber, R., Bannasch, D. L., & Brennan, P. (2011). *Aggression*. Amsterdam, the Netherlands: Academic.

Huesmann, R. L. (1988). An information processing model for the development of aggression. *Aggressive Behavior, 14*(1), 13-24. doi:10.1002/1098-2337(1988)14:1<13::AID-AB2480140104>3.0.CO;2-J

Humphreys, K. (2017). A review of the impact of exclusion criteria on the generalizability of schizophrenia treatment research. *Clinical Schizophrenia & Related Psychoses, 11*(1), 49-57. doi:10.3371/1935-1232-11.1.49

Huss, M. T. (2009). *Forensic psychology: Research, practice, and applications*. Malden, MA: Blackwell Pub.

Im, D. S. (2016). Template to perpetrate: An update on violence in autism spectrum disorder. *Harvard Review of Psychiatry, 24*(1), 14-35. doi:10.1097/HRP.0000000000000087

Iofrida, C., Palumbo, S., & Pellegrini, S. (2014). Molecular genetics and antisocial behavior: where do we stand? *Experimental Biology and Medicine, 239*(11), 1514-1523. doi:10.1177/1535370214529508

Iozzino, L., Ferrari, C., Large, M., Nielssen, O., & de Girolamo, G. (2015). Prevalence and risk factors of violence by psychiatric acute inpatients: A systematic review and meta-analysis. *PloS One, 10*(6), e0128536. doi:10.1371/journal.pone.0128536

Jacobs, P. A., Brunton, M., Melville, M. M., Brittain, R. P., & McClemont, W. F. (1965). Aggressive behavior, mental sub-normality and the XYY male. *Nature, 208*(5017), 1351-1352.

Jakobsson, U., & Westergren, A. (2005). Statistical methods for assessing agreement for ordinal data. *Scandinavian Journal of Caring Sciences, 19*(4), 427-431. doi:10.1111/j.1471-6712.2005.00368.x

Jennings, W. G., Piquero, A. R., & Reingle, J. M. (2012). On the overlap between victimization and offending: A review of the literature. *Aggression and Violent Behavior, 17*(1), 16-26. doi:10.1016/j.avb.2011.09.003

Jennings, W. G., & Reingle, J. M. (2014). Violent Victimization. In J. Mitchell Miller (Ed.), *The encyclopedia of theoretical criminology*. Chichester, UK: Wiley Blackwell.

Joseph, J. (2001). Is crime in the genes? A critical review of twin and adoption studies of criminality and antisocial behavior. *The Journal of Mind and Behavior, 22*(2), 179-218.

Kattan, M. W., & Cowen, M. E. (2009). *Encyclopedia of medical decision making*. Thousand Oaks, CA: SAGE Publications.

Katz, M. H. (2006). *Study design and statistical analysis: A practical guide for clinicians*. Cambridge, UK: Cambridge University Press.

Kendler, K. S., Larsson Lönn, S., Morris, N. A., Sundquist, J., Långström, N., & Sundquist, K. (2014). A Swedish national adoption study of criminality. *Psychological Medicine, 44*(9), 1913-1925. doi:10.1017/S0033291713002638

Kim, E. (2004). Neurobehavioral aspects of aggression. In James P. Morgan (Ed.), *Focus on aggression research*. Hauppauge, NY: Nova Science Publishers.

King, G., & Nielsen, R. (2016). Working paper. *Why propensity scores should not be used for matching*. Copy at http://j.mp/2ovYGsW.

Kjellström, J. (1909). *Hälsingelagen tolkad*. Uppsala, Sweden: Uppsala University.

Klepfisz, G., Daffern, M., & Day, A. (2016). Understanding dynamic risk factors for violence. *Psychology, Crime & Law, 22*(1-2), 124-137. doi:10.1080/1068316X.2015.1109091

Klötz, F., Petersson, A., Isacson, D., & Thiblin, I. (2007). Violent crime and substance abuse: a medico-legal comparison between deceased users of anabolic androgenic steroids and abusers of illicit drugs. *Forensic Science International, 173*(1), 57-63. doi:10.1016/j.forsciint.2007.01.026

Knutsson, J. (1977). *Labeling theory: A critical examination*. Stockholm, Sweden: National Swedish council for crime prevention, Liber distribution.

Koch, G. (2015). *Basic allied health statistics and analysis* (4th ed.). Stamford, CT: Cengage Learning.

Koo, T. K., & Li, M. Y. (2016). A guideline of selecting and reporting intraclass correlation coefficients for reliability research. *Journal of Chiropractic Medicine, 15*(2), 155-163. doi:10.1016/j.jcm.2016.02.012

Korneva, E. A., & Phelps, C. (2008). *Cytokines and the brain*. Oxford, UK: Elsevier.

Kraemer, H. C., Kazdin, A. E., Offord, D. R., Kessler, R. C., Jensen, P. S., & Kupfer, D. J. (1997). Coming to terms with the terms of risk. *Archives of General Psychiatry, 54*(4), 337-343.

Kraepelin, E. (1904). *Psychiatrie: Ein Lehrbuch für Studierende und Arzte*. Leipzig, Germany: Verlag von Johann Ambrosius Barth.

Kropp, P. R. (1995). *Manual for the spousal assault risk assessment guide* (2nd ed.). Vancouver, BC: The British Columbia Institute on Family Violence.

Kurtz, L. R., & Turpin, J. E. (1999). *Encyclopedia of violence, peace & conflict*. San Diego, CA: Academic Press.

Lansford, J. E., Miller-Johnson, S., Berlin, L. J., Dodge, K. A., Bates, J. E., & Pettit, G. S. (2007). Early physical abuse and later violent delinquency: A prospective longitudinal study. *Child Maltreatment, 12*(3), 233-245. doi:10.1177/1077559507301841

LaPrairie, J. L., Schechter, J. C., Robinson, B. A., & Brennan, P. A. (2011). Perinatal risk factors in the development of aggression and violence. *Advances in Genetics, 75*, 215-253. doi:10.1016/B978-0-12-380858-5.00004-6

Large, M. M., & Nielssen, O. (2011). Violence in first-episode psychosis: A systematic review and meta-analysis. *Schizophrenia Research, 125*(2-3), 209-220. doi:10.1016/j.schres.2010.11.026

Latalova, K., Kamaradova, D., & Prasko, J. (2014). Violent victimization of adult patients with severe mental illness: A systematic review. *Neuropsychiatric Disease and Treatment, 10*, 1925-1939. doi:10.2147/NDT.S68321

Laucht, M., Brandeis, D., & Zohsel, K. (2014). Gene-environment interactions in the etiology of human violence. *Current Topics in Behavioral Neurosciences, 17*, 267-295. doi:10.1007/7854_2013_260

Lee, R. (2015). The psychofarmacology of criminality and oxytocin modulation of social behavior in aggressive individuals. In Matt DeLisi & Michael G Vaughn (Eds.), *The Routledge international handbook of biosocial criminology*. London, UK: Routledge, Taylor & Francis Group.

Lidz, C. W., Mulvey, E. P., & Gardner, W. (1993). The accuracy of predictions of violence to others. *JAMA, 269*(8), 1007-1011. doi:10.1001/jama.269.8.1007

Lilly, J. R., Cullen, F. T., & Ball, R. A. (2015). *Criminological theory: Context and consequences* (6th ed.). Thousand Oaks, CA: SAGE Publications.

Lissitz, R. W., & Samuelsen, K. (2007). A suggested change in terminology and emphasis regarding validity and education. *Educational Researcher, 36*(8), 437-448. doi:10.3102/0013189X07311286

Locke, J. (1988). Paternal power. In Peter Laslett (Ed.), *Two treatises of government*. Cambridge, UK: Cambridge University Press.

Lösel, F., & Farrington, D. P. (2012). Direct protective and buffering protective factors in the development of youth violence. *American Journal of Preventive Medicine, 43*(2 Suppl 1), S8-S23. doi:10.1016/j.amepre.2012.04.029

Maclean, F., & Beak, K. (2012). *Factors associated with serious or persistent violent offending: Findings from a rapid evidence assessment*. Retrieved from: http://whatworks.college.police.uk/Research/Documents/REA_violent_reoffending.pdf

Maguire, M., Morgan, R., & Reiner, R. (2012). *The Oxford handbook of criminology* (5th ed.). Oxford, UK: Oxford University Press.

Maniglio, R. (2009). Severe mental illness and criminal victimization: A systematic review. *Acta Psychiatrica Scandinavica, 119*(3), 180-191. doi:10.1111/j.1600-0447.2008.01300.x

Manu. (1991). *The Laws of Manu: With an introduction and notes*. London, UK: Penguin.

Martínez-Mesa, J., González-Chica, D. A., Duquia, R. P., Bonamigo, R. R., & Bastos, J. L. (2016). Sampling: How to select participants in my research study? *Anais Brasileiros de Dermatologia, 91*(3), 326-330. doi:10.1590/abd1806-4841.20165254

Matsueda, R. L., Kreager, D. A., & Huizinga, D. (2006). Deterring delinquents: A rational choice model of theft and violence. *American Sociological Review, 71*(1), 95-122. doi:10.1177/000312240607100105

Matthies, S., Rüsch, N., Weber, M., Lieb, K., Philipsen, A., Tuescher, O., . . . van Elst, L. T. (2012). Small amygdala-high aggression? The role of the amygdala in modulating aggression in healthy subjects. *World Journal of Biological Psychiatry, 13*(1), 75-81. doi:10.3109/15622975.2010.541282

Maxim, L. D., Niebo, R., & Utell, M. J. (2014). Screening tests: A review with examples. *Inhalation Toxicology, 26*(13), 811-828. doi:10.3109/08958378.2014.955932

Mazur, A., & Booth, A. (1998). Testosterone and dominance in men. *Behavioral and Brain Sciences, 21*(3), 353-363; discussion 363-397.

McDermott, B. E., Dualan, I. V., & Scott, C. L. (2011). The predictive ability of the Classification of Violence Risk (COVR) in a forensic psychiatric hospital. *Psychiatric Services, 62*(4), 430-433. doi:10.1176/ps.62.4.pss6204_0430

McDonald, P. (2004). *Oxford dictionary of medical quotations*. Oxford, UK: Oxford University Press.

McGraw, K. O., & Wong, S. P. (1992). A common language effect size statistic. *Psychological Bulletin, 111*(2), 361-365.

McHugh, M. L. (2012). Interrater reliability: The kappa statistic. *Biochemia Medica: Casopis Hrvatskoga Drustva Medicinskih Biokemicara, 22*(3), 276-282.

Mechoulam, R. (2002). Discovery of endocannabinoids and some random thoughts on their possible roles in neuroprotection and aggression. *Prostaglandins, Leukotrienes, and Essential Fatty Acids, 66*(2-3), 93-99. doi:10.1054/plef.2001.0340

Mednick, S. A., Gabrielli, W. F., & Hutchings, B. (1984). Genetic influences in criminal convictions: Evidence from an adoption cohort. *Science, 224*(4651), 891-894.

Meldrum, B. S. (2000). Glutamate as a neurotransmitter in the brain: Review of physiology and pathology. *Journal of Nutrition, 130*(4S Suppl), 1007S-1015S.

Menkes, D. B., & Herxheimer, A. (2015). Depression and violence—what do we really know? *Lancet Psychiatry, 2*(6), 491-492. doi:10.1016/S2215-0366(15)00166-2

Merton, R. K. (1938). Social structure and anomie. *American Sociological Review, 3*(5), 672-682. doi:10.2307/2084686

Miczek, K. A., DeBold, J. F., Hwa, L. S., Newman, E. L., & de Almeida, R. M. (2015). Alcohol and violence: Neuropeptidergic modulation of monoamine systems. *Annals of the New York Academy of Sciences, 1349*, 96-118. doi:10.1111/nyas.12862

Miczek, K. A., Fish, E. W., De Almeida, R. M., Faccidomo, S., & Debold, J. F. (2004). Role of alcohol consumption in escalation to violence. *Annals of the New York Academy of Sciences, 1036*, 278-289. doi:10.1196/annals.1330.018

Miczek, K. A. (1987). The psychopharmacology of aggression. In Leslie L Iversen, Susan D Iversen, & Solomon H Snyder (Eds.), *Handbook of psychopharmacology. Vol.19, New directions in behavioral pharmacology*. New York, NY: Plenum Press.

Miczek, K. A., DeBold, J. F., Haney, M., Tidey, J., Jeffrey, V., & Weerts, E. M. (1994). Alcohol, drugs of abuse, aggression, and violence. In Albert J Reiss & Jeffrey A Roth (Eds.), *Understanding and preventing violence, volume 3: Social influences*. Washington, DC: National Academies Press.

Miczek, K. A., & Fish, E. W. (2006). Monoamines, GABA, glutamate, and aggression. In Randy Joe Nelson (Ed.), *Biology of aggression*. Oxford, UK: Oxford University Press.

Miles, D. R., & Carey, G. (1997). Genetic and environmental architecture of human aggression. *Journal of Personality and Social Psychology, 72*(1), 207-217.

Miles, S. H. (2004). *The Hippocratic Oath and the ethics of medicine*. Oxford, UK: Oxford University Press.

Miller, J. M. (2009). *21st century criminology: A reference handbook*. Thousand Oaks, CA: SAGE Publications.

Minh, A., Matheson, F. I., Daoud, N., Hamilton-Wright, S., Pedersen, C., Borenstein, H., & O'Campo, P. (2013). Linking childhood and adult criminality: Using a life course framework to examine childhood abuse and neglect, substance use and adult partner violence. *International Journal of Environmental Research and Public Health, 10*(11), 5470-5489. doi:10.3390/ijerph10115470

Mirzakhanian, H., Sanchez, S., Addington, J., Bearden, C., Cannon, T., Cornblatt, B., . . . Cadenhead, K. (2017). Risk of violence in attenuated psychosis symptomos syndrome and its relationship with symptology. *Schizophrenia Bulletin, 43*(suppl_1), S106-S107. doi:10.1093/schbul/sbx021.287

Moberg, T., Stenbacka, M., Jönsson, E. G., Nordström, P., Asberg, M., & Jokinen, J. (2014). Risk factors for adult interpersonal violence in suicide attempters. *BMC Psychiatry, 14*, 195. doi:10.1186/1471-244X-14-195

Monahan, J., & Skeem, J. L. (2014). The evolution of violence risk assessment. *CNS Spectrums, 19*(5), 419-424. doi:10.1017/S1092852914000145

Monahan, J., Steadman, H. J., Appelbaum, P., Grisso, T., Mulvey, E., Roth, L., . . . Silver, E. (2005). COVR^{TM}. The classification of violence risk. Lutz, FA: Psychological Assessment Resources.

Monahan, J., Steadman, H. J., Appelbaum, P. S., Grisso, T., Mulvey, E. P., Roth, L. H., . . . Silver, E. (2006). The classification of violence risk. *Behavioral Sciences and the Law, 24*(6), 721-730. doi:10.1002/bsl.725

Monahan, J., Steadman, H. J., Appelbaum, P. S., Robbins, P. C., Mulvey, E. P., Silver, E., . . . Grisso, T. (2000). Developing a clinically useful actuarial tool for assessing violence risk. *The British Journal of Psychiatry, 176*, 312-319. doi:10.1192/bjp.176.4.312

Monahan, J., Steadman, H. J., Silver, E., Appelbaum, P. S., Clark Robbins, P., Mulvey, E. P., . . . Banks, S. (2001). *Rethinking risk assessment: The MacArthur study of mental disorder and violence*. New York, NY: Oxford University Press.

Monro, C. H. (1904). *The digest of Justinian. Translated by C. H. Monro. Volume 1.* Cambridge, UK: University Press.

Morrison, W. (1995). *Theoretical criminology: From modernity to post-modernism*. London, UK: Cavendish.

Morton, W. A. (1999). Cocaine and Psychiatric Symptoms. *Primary Care Companion to the Journal of Clinical Psychiatry, 1*(4), 109-113.

Mueser, K. T., Goodman, L. B., Trumbetta, S. L., Rosenberg, S. D., Osher, F. C., Vidaver, R., . . . Foy, D. W. (1998). Trauma and posttraumatic stress disorder in severe mental illness. *Journal of Consulting and Clinical Psychology, 66*(3), 493-499.

Munktell, H. (1943). Straffrätt och sinnessjuka. Några historiska data. *Svensk Juristtidning, 1*, 111-118.

Murray, A. L., Eisner, M., Obsuth, I., & Ribeaud, D. (2017). Situating violent ideations within the landscape of mental health: Associations between violent ideations and dimensions of mental health. *Psychiatry Research, 249*, 70-77. doi:10.1016/j.psychres.2017.01.005

Möller, A., & Hell, D. (2002). Eugen Bleuler and forensic psychiatry. *International Journal of Law and Psychiatry, 25*(4), 351-360.

Nagtegaal, M. H., Rassin, E., & Muris, P. (2006). Aggressive fantasies, thought control strategies, and their connection to aggressive behaviour. *Personality and Individual Differences, 41*(8), 1397-1407. doi:10.1016/j.paid.2006.05.009

Narvaes, R., & de Almeida, R. M. M. (2014). Aggressive behavior and three neurotransmitters: Dopamine, GABA, and serotonin—a review of the last 10 years. *Psychology & Neuroscience, 7*(4), 601-607. doi:10.3922/j.psns.2014.4.20

National Board of Forensic Medicine. (2017). *Årsredovisning 2016*. Retrieved from https://www.rmv.se/wp-content/uploads/%C3%85rsredovisning-RMV-2016.pdf

National Collaborating Centre for Mental Health. (2010). *Antisocial personality disorder: Treatment, management and prevention*. London, UK: British Psychological Society.

National Collaborating Centre for Mental Health. (2015). *Violence and aggression: Short-Term management in mental health, health and community settings: Updated edition.* Retrieved from https://www.ncbi.nlm.nih.gov/pubmedhealth/PMH0078691/pdf/PubMedHealth_PMH0078691.pdf

National Council for Crime Prevention. (2017). *Kriminalstatistik 2016: Personer lagförda för brott, slutlig statistik*. Retrieved from https://www.bra.se/download/18.62fc8fb415c2ea106932e8e/1496215844157/Sammanfattning_lagforda_2016.pdf

Nationellt rättspsykiatriskt kvalitetsregister. (2017). *Årsrapport 2016*. Retrieved from http://www.psykiatriregister.se/sites/default/files/documents/pdfversion2_rattspsyk_2016.pdf

Newman, E. L., Smith, K. S., Takahashi, A., Chu, A., Hwa, L. S., Chen, Y., . . . Miczek, K. A. (2015). α2-containing GABA(A) receptors: A requirement for midazolam-escalated aggression and social approach in mice. *Psychopharmacology, 232*(23), 4359-4369. doi:10.1007/s00213-015-4069-9

Neylan, T. C. (1999). Frontal lobe function: Mr. Phineas Gage's famous injury. *Journal of Neuropsychiatry and Clinical Neurosciences, 11*(2), 280-281. doi:10.1176/jnp.11.2.280

Norström, T., & Rossow, I. (2014). Cannabis use and violence: Is there a link? *Scandinavian Journal of Public Health, 42*(4), 358-363. doi:10.1177/1403494814525003

Nuss, P. (2015). Anxiety disorders and GABA neurotransmission: A disturbance of modulation. *Neuropsychiatric Disease and Treatment, 11*, 165-175. doi:10.2147/NDT.S58841

O'Reilly, K., Donohoe, G., Coyle, C., O'Sullivan, D., Rowe, A., Losty, M., . . . Kennedy, H. G. (2015). Prospective cohort study of the relationship between neuro-cognition, social cognition and violence in forensic patients with schizophrenia and schizoaffective disorder. *BMC Psychiatry, 15*, 155. doi:10.1186/s12888-015-0548-0

O'Shea, L. E., & Dickens, G. L. (2014). Short-Term Assessment of Risk and Treatability (START): Systematic review and meta-analysis. *Psychological Assessment, 26*(3), 990-1002. doi:10.1037/a0036794

O'Shea, L. E., & Dickens, G. L. (2016). Performance of protective factors assessment in risk prediction for adults: Systematic review and meta-analysis. *Clinical Psychology: Science and Practice, 23*(2), 126-138. doi:10.1111/cpsp.12146

Oberlander, J. G., & Henderson, L. P. (2012). The Sturm und Drang of anabolic steroid use: Angst, anxiety, and aggression. *Trends in Neurosciences, 35*(6), 382-392. doi:10.1016/j.tins.2012.03.001

Office of the Surgeon General. (2001). *Youth violence: A report of the Surgeon General*. Washington, DC: Office of the Surgeon General.

Ojala, K. P., Tiihonen, J., Repo-Tiihonen, E., Tikkanen, R., & Virkkunen, M. (2015). Basal insulin secretion, PCL-R and recidivism among impulsive violent alcoholic offenders. *Psychiatry Research, 225*(3), 420-424. doi:10.1016/j.psychres.2014.11.073

Okasha, A. (2003). The declaration of Madrid and its implementation. An update. *World Psychiatry, 2*(2), 65-67.

Okoli, G. N., Sanders, R. D., & Myles, P. (2014). Demystifying propensity scores. *British Journal of Anaesthesia, 112*(1), 13-15. doi:10.1093/bja/aet290

Oram, S., Trevillion, K., Khalifeh, H., Feder, G., & Howard, L. M. (2014). Systematic review and meta-analysis of psychiatric disorder and the perpetration of partner violence. *Epidemiology and Psychiatric Sciences, 23*(4), 361-376. doi:10.1017/S2045796013000450

Otto, R. K., & Douglas, K. S. (2010). *Handbook of violence risk assessment*. New York, NY: Routledge.

Page, K. (2012). The four principles: Can they be measured and do they predict ethical decision making? *BMC Medical Ethics, 13*, 10. doi:10.1186/1472-6939-13-10

Pardini, D. A., Raine, A., Erickson, K., & Loeber, R. (2014). Lower amygdala volume in men is associated with childhood aggression, early psychopathic traits, and future violence. *Biological Psychiatry, 75*(1), 73-80. doi:10.1016/j.biopsych.2013.04.003

Pavlov, K. A., Chistiakov, D. A., & Chekhonin, V. P. (2012). Genetic determinants of aggression and impulsivity in humans. *Journal of Applied Genetics, 53*(1), 61-82. doi:10.1007/s13353-011-0069-6

Pearce, N. (2016). Analysis of matched case-control studies. *BMJ, 352*, 969.

Pedersen, C. A. (2013). Oxytocin regulation of maternal behavior: From rodents to humans. In Elena Choleris, Donald W Pfaff, & Martin Kavaliers (Eds.), *Oxytocin, vasopressin, and related peptides in the regulation of behavior*. Cambridge, UK: Cambridge University Press.

Peterson, J. K., Skeem, J., Kennealy, P., Bray, B., & Zvonkovic, A. (2014). How often and how consistently do symptoms directly precede criminal behavior among offenders with mental illness? *Law and Human Behavior, 38*(5), 439-449. doi:10.1037/lhb0000075

Plato & Pangle, T. L. (1988). *The laws of Plato* (University of Chicago Press ed.). Chicago, IL: University of Chicago Press.

Pollock, V. E., Briere, J., Schneider, L., Knop, J., Mednick, S. A., & Goodwin, D. W. (1990). Childhood antecedents of antisocial behavior: Parental alcoholism and physical abusiveness. *American Journal of Psychiatry, 147*(10), 1290-1293. doi:10.1176/ajp.147.10.1290

Porta, M. S., Greenland, S., & Last, J. M. (2008). *A dictionary of epidemiology* (5th ed.). Oxford, UK: Oxford University Press.

Pratt, T. C., Cullen, F. T., Sellers, C. S., Winfree, L. T., Madensen, T. D., Daigle, L. E., . . . Gau, J. M. (2010). The empirical status of social learning theory: A meta-analysis. *Justice Quarterly, 27*(6), 765-802. doi:10.1080/07418820903379610

Preedy, V. R. editor. (2017). *The Neuroscience of cocaine: Mechanisms and treatment*. Cambridge, MA: Academic Press.

Qvarsell, R. (1993). *Utan vett och vilja: Om synen på brottslighet och sinnessjukdom*. Stockholm, Sweden: Carlssons Bokförlag.

Ray, L. J. (1999). *Theorizing classical sociology*. Buckingham, UK: Open University Press.

Reif, A., Rösler, M., Freitag, C. M., Schneider, M., Eujen, A., Kissling, C., . . . Retz, W. (2007). Nature and nurture predispose to violent behavior: Serotonergic genes and adverse childhood environment. *Neuropsychopharmacology, 32*(11), 2375-2383. doi:10.1038/sj.npp.1301359

Reiss, A. J., & Roth, J. A. (1993). *Understanding and preventing violence*. Washington, DC: National Academy Press.

Renwick, L., Stewart, D., Richardson, M., Lavelle, M., James, K., Hardy, C., . . . Bowers, L. (2016). Aggression on inpatient units: Clinical characteristics and consequences. *International Journal of Mental Health Nursing, 25*(4), 308-318. doi:10.1111/inm.12191

Renzetti, C. M., & Edleson, J. L. (2008). *Encyclopedia of interpersonal violence*. Thousand Oaks, CA: SAGE Publications.

Retz, W., & Rösler, M. (2009). The relation of ADHD and violent aggression: What can we learn from epidemiological and genetic studies? *International Journal of Law and Psychiatry, 32*(4), 235-243. doi:10.1016/j.ijlp.2009.04.006

Rice, M. E., & Harris, G. T. (1995). Violent recidivism: Assessing predictive validity. *Journal of Consulting and Clinical Psychology, 63*(5), 737-748.

Riegelman, R. K. (2013). *Studying a study & testing a test: Reading evidence-based health research* (6th ed.). Philadelphia, PA: Wolters Kluwer/Lippincott Williams & Wilkins Heath.

Robinson, D. N. (2013). The insanity defense as a history of mental disorder. In K. W. M. Fulford (Ed.), *The Oxford handbook of philosophy and psychiatry*. Oxford, UK: Oxford University Press.

Rose, S., & Laan, M. J. (2009). Why match? Investigating matched case-control study designs with causal effect estimation. *The International Journal of Biostatistics, 5*(1), Article 1. doi:10.2202/1557-4679.1127

Rossegger, A., Endrass, J., Gerth, J., & Singh, J. P. (2014). Replicating the violence risk appraisal guide: A total forensic cohort study. *PloS One, 9*(3), e91845. doi:10.1371/journal.pone.0091845

Rothwell, P. M. (2007). *The Lancet: Treating individuals: From randomised trials to personalised medicine*. New York, NY: Elsevier.

Rueve, M. E., & Welton, R. S. (2008). Violence and mental illness. *Psychiatry (Edgmont), 5*(5), 34-48.

Sadeh, N., Binder, R. L., & McNiel, D. E. (2014). Recent victimization increases risk for violence in justice-involved persons with mental illness. *Law and Human Behavior, 38*(2), 119-125. doi:10.1037/lhb0000043

Sahara, S., Yanagawa, Y., O'Leary, D. D., & Stevens, C. F. (2012). The fraction of cortical GABAergic neurons is constant from near the start of cortical neurogenesis to adulthood. *Journal of Neuroscience, 32*(14), 4755-4761. doi:10.1523/JNEUROSCI.6412-11.2012

Salkind, N. J. (2010). *Encyclopedia of research design*. Thousand Oaks, CA: SAGE Publications.

Sariaslan, A., Lichtenstein, P., Larsson, H., & Fazel, S. (2016). Triggers for Violent Criminality in Patients With Psychotic Disorders. *JAMA Psychiatry, 73*(8), 796-803. doi:10.1001/jamapsychiatry.2016.1349

Sawilowsky, S. S. (2009). New effect size rules of thumb. *Journal of Modern Applied Statistical Methods, 8*(2), 597-599.

Schachter, D., Gilbert, D., Wegner, D., & Hood, B. (2016). *Psychology: Second European edition*. London, UK: Macmillan Publishers Ltd.

Schoeler, T., Theobald, D., Pingault, J. B., Farrington, D. P., Jennings, W. G., Piquero, A. R., . . . Bhattacharyya, S. (2016). Continuity of cannabis use and violent offending over the life course. *Psychological Medicine, 46*(8), 1663-1677. doi:10.1017/S0033291715003001

Schuster, D. P., & Powers, W. J. M. D. (2005). *Translational and experimental clinical research*. Philadelphia, PA: Lippincott Williams & Wilkins.

Schwartz, D., Dodge, K. A., & Coie, J. D. (1993). The emergence of chronic peer victimization in boys' play groups. *Child Development, 64*(6), 1755-1772.

Scurich, N. (2016). An introduction to the assessment of violence risk. In Jay P Sing, Stål Bjørkly, & Seena Fazel (Eds.), *International perspectives on violence risk assessment*. New York, NY: Oxford University Press.

Sheldon, K., Davies, J., & Howells, K. (2011). *Research in practice for forensic professionals*. New York, NY: Routledge.

Shields, I. W. (1990). *Young Offender-Level of Service Inventory, manual and norms*. Ottawa, ON: Ontario Ministry of Correctional Services.

Siever, L. J. (2008). Neurobiology of aggression and violence. *American Journal of Psychiatry, 165*(4), 429-442. doi:10.1176/appi.ajp.2008.07111774

Silver, E., Piquero, A. R., Jennings, W. G., Piquero, N. L., & Leiber, M. (2011). Assessing the violent offending and violent victimization overlap among discharged psychiatric patients. *Law and Human Behavior, 35*(1), 49-59. doi:10.1007/s10979-009-9206-8

Sim, J., & Wright, C. C. (2000). *Research in health care: Concepts, designs and methods*. Cheltenham, UK: Stanley Thornes Ltd.

Sinai, C., Hirvikoski, T., Nordström, A. L., Nordström, P., Nilsonne, Å, Wilczek, A., . . . Jokinen, J. (2015). Thyroid hormones and adult interpersonal violence among women with borderline personality disorder. *Psychiatry Research, 227*(2-3), 253-257. doi:10.1016/j.psychres.2015.03.025

Singh, J. P., Desmarais, S. L., & Van Dorn, R. A. (2013). Measurement of predictive validity in violence risk assessment studies: A second-order systematic review. *Behavioral Sciences and the Law, 31*(1), 55-73. doi:10.1002/bsl.2053

Singh, J. P., Desmarais, S. L., Hurducas, C., Arbach-Lucioni, K., Condemarin, C., Dean, K., . . . Otto, R. K. (2014). International perspectives on the practical application of violence risk assessment: A global survey of 44 countries. *International Journal of Forensic Mental Health, 13*(**3**), 193-206. doi:10.1080/14999013.2014.922141

Singh, J. P., Grann, M., & Fazel, S. (2011). A comparative study of violence risk assessment tools: A systematic review and metaregression analysis of 68 studies involving 25,980 participants. *Clinical Psychology Review, 31*(3), 499-513. doi:10.1016/j.cpr.2010.11.009

Singh, J. P., Serper, M., Reinharth, J., & Fazel, S. (2011). Structured assessment of violence risk in schizophrenia and other psychiatric disorders: A systematic review of the validity, reliability, and item content of 10 available instruments. *Schizophrenia Bulletin, 37*(5), 899-912. doi:10.1093/schbul/sbr093

Smuk, M., Carpenter, J. R., & Morris, T. P. (2017). What impact do assumptions about missing data have on conclusions? A practical sensitivity analysis for a cancer survival registry. *BMC Medical Research Methodology, 17*(1), 21. doi:10.1186/s12874-017-0301-0

Snowden, R. J., Gray, N. S., Taylor, J., & Fitzgerald, S. (2009). Assessing risk of future violence among forensic psychiatric inpatients with the Classification of Violence Risk (COVR). *Psychiatric Services, 60*(11), 1522-1526. doi:10.1176/appi.ps.60.11.1522

Spatz Widom, C. (1989). Child abuse, neglect, and violent criminal behavior. *Criminology, 27*(2), 251 271. doi:10.1111/j.1745-9125.1989.tb01032.x

Sperry, L. (2016). *Mental health and mental disorders: An encyclopedia of conditions, treatments, and well-being*. Santa Barbara, CA: Greenwood.

Stalenheim, E. G. (2004). Long-term validity of biological markers of psychopathy and criminal recidivism: Follow-up 6-8 years after forensic psychiatric investigation. *Psychiatry Research, 121*(3), 281-291.

Stenbacka, M., Moberg, T., Romelsjö, A., & Jokinen, J. (2012). Mortality and causes of death among violent offenders and victims--a Swedish population based longitudinal study. *BMC Public Health, 12*, 38. doi:10.1186/1471-2458-12-38

Sterne, J. A., White, I. R., Carlin, J. B., Spratt, M., Royston, P., Kenward, M. G., . . . Carpenter, J. R. (2009). Multiple imputation for missing data in epidemiological and clinical research: Potential and pitfalls. *BMJ, 338*, b2393.

Stevenson, H. E., & Wormith, J. S. (1987). *Psychopathy and the Level of Service Inventory*. Ottawa, ON: Ministry of the Solicitor General of Canada.

Strub, D. S., Douglas, K. S., & Nicholls, T. L. (2014). The validity of version 3 of the HCR-20 violence risk assessment scheme amongst offenders and civil psychiatric patients. *International Journal of Forensic Mental Health, 13*(2), 148-159. doi:10.1080/14999013.2014.911785

Stuart, E. A. (2010). Matching methods for causal inference: A review and a look forward. *Statistical Science, 25*(1), 1-21. doi:10.1214/09-STS313

Studer, E., Näslund, J., Westman, A., Carlsson, A., & Eriksson, E. (2016). The effects of the dopamine stabilizer (-)-OSU6162 on aggressive and sexual behavior in rodents. *Translational Psychiatry, 6*, e762. doi:10.1038/tp.2016.12

Sturup, J., Monahan, J., & Kristiansson, M. (2013). Violent behavior and gender of Swedish psychiatric patients: A prospective clinical study. *Psychiatric Services, 64*(7), 688-693. doi:10.1176/appi.ps.201200266

Suarez, E. C., Lewis, J. G., & Kuhn, C. (2002). The relation of aggression, hostility, and anger to lipopolysaccharide-stimulated tumor necrosis factor (TNF)-alpha by blood monocytes from normal men. *Brain, Behavior, and Immunity, 16*(6), 675-684.

Sutherland, E. H. (1939). *Principles of criminology*. Chicago, IL: J. B. Lippincott company.

Swedish Association of Local Authorities and Regions. (2017). *Verksamhetstabeller 2016*. In TabellbilagaV2016, Excel. Swedish Association of Local Authorities and Regions.

Swedish Penal Code. (1962). *Swedish Code of Statutes*.

Svennerlind, C., Nilsson, T., Kerekes, N., Andiné, P., Lagerkvist, M., Forsman, A., . . . Malmgren, H. (2010). Mentally disordered criminal offenders in the Swedish criminal system. *International Journal of Law and Psychiatry, 33*(4), 220-226. doi:10.1016/j.ijlp.2010.06.003

Teplin, L. A., McClelland, G. M., Abram, K. M., & Weiner, D. A. (2005). Crime victimization in adults with severe mental illness: Comparison with the National Crime Victimization Survey. *Archives of General Psychiatry, 62*(8), 911-921. doi:10.1001/archpsyc.62.8.911

Thiblin, I., & Pärlklo, T. (2002). Anabolic androgenic steroids and violence. *Acta Psychiatrica Scandinavica. Supplementum*(412), 125-128.

Tierney, J. J. (2009). *Key perspectives in criminology*. Maidenhead, UK: Open University Press.

Tiihonen, J., Rautiainen, M. R., Ollila, H. M., Repo-Tiihonen, E., Virkkunen, M., Palotie, A., . . . Paunio, T. (2015). Genetic background of extreme violent behavior. *Molecular Psychiatry, 20*(6), 786-792. doi:10.1038/mp.2014.130

Tremblay, R. E., Hartup, W. W., & Archer, J. (2005). *Developmental origins of aggression*. New York, NY: Guilford Press.

Ttofi, M. M., Farrington, D. P., Piquero, A. R., Lösel, F., DeLisi, M., & Murray, J. (2016). Intelligence as a protective factor against offending: A meta-analytic review of prospective longitudinal studies. *Journal of Criminal Justice, 45*, 4-18. doi:10.1016/j.jcrimjus.2016.02.003

Tuvblad, C., & Baker, L. A. (2011). Human aggression across the lifespan: Genetic propensities and environmental moderators. *Advances in Genetics, 75*, 171-214. doi:10.1016/B978-0-12-380858-5.00007-1

United Nations Office on Drugs and Crime. (2013). *Global study on homicide 2013*. Vienna, Austria: United Nations Publications.

Walker, K., Bowen, E., & Brown, S.. (2013). Psychological and criminological factors associated with desistance from violence: A review of the literature. *Aggression and Violent Behavior, 18*(2), 286-299. doi:10.1016/j.avb.2012.11.021

Wang, P. S., Lane, M., Olfson, M., Pincus, H. A., Wells, K. B., & Kessler, R. C. (2005). Twelve-month use of mental health services in the United States: Results from the national comorbidity survey replication. *Archives of General Psychiatry, 62*(6), 629-640. doi:10.1001/archpsyc.62.6.629

Ward, T. (2016). Dynamic risk factors: Scientific kinds of predictive constructs. *Psychology, Crime & Law, 22*(1-2), 2-16. doi:10.1080/1068316X.2015.1109094

Ward, T., & Beech, A. R. (2015). Dynamic risk factors: A theoretical dead-end? *Psychology, Crime & Law, 21*(2), 100-113. doi:10.1080/1068316X.2014.917854

Ward, T., & Maruna, S.. (2007). *Rehabilitation: Beyond the risk paradigm*. London, UK: Routledge.

Varshney, M., Mahapatra, A., Krishnan, V., Gupta, R., & Deb, K. S. (2016). Violence and mental illness: What is the true story? *Journal of Epidemiology and Community Health, 70*(3), 223-225. doi:10.1136/jech-2015-205546

Watt, B. D., Kohphet, A., Oberin, D., & Keating, S. (2013). The relationship between violent fantasy and alcohol misuse in aggressive behaviours. *Australian Psychologist, 48*(6), 452-458. doi:10.1111/ap.12011

Vaughn, M. G., DeLisi, M., Beaver, K. M., & Wright, J. P. (2009). DAT1 and 5HTT are associated with pathological criminal behavior in a nationally representative sample of youth. *Criminal Justice and Behavior, 36*(11), 1113-1124. doi:10.1177/0093854809342839

Webster, C. D., Eaves, D., Douglas, K. S., & Wintrup, A. M. (1995). *The HCR-20 scheme: The assessment of dangerousness and risk (Version 1)*. Burnaby, BC: Simon Fraser University.

Webster, C. D., Martin, M., Brink, J., Nicholls, T. L, & Desmarais, S. L. (2009). *Manual for the Short Term Assessment of Risk and Treatability (START). Version 1.1*. Port Coquitlam, BC: BC Mental Health and Addiction Services.

Webster, C. D., Douglas, K. S., Eaves, D., & Hart, S. D. (1997). *HCR-20: Assessing risk for violence, version 2*. Burnaby, BC: Simon Fraser University.

Wenzel, S. L., Tucker, J. S., Elliott, M. N., Marshall, G. N., & Williamson, S. L. (2004). Physical violence against impoverished women: A longitudinal analysis of risk and protective factors. *Women's Health Issues, 14*(5), 144-154. doi:10.1016/j.whi.2004.06.001

Wetzell, R. F. (2000). *Inventing the criminal: A history of German criminology, 1880-1945*. Chapel Hill, NC: University of North Carolina Press.

Wharton, T. C., & Ford, B. K. (2014). What is known about dementia care recipient violence and aggression against caregivers? *Journal of Gerontological Social Work, 57*(5), 460-477. doi:10.1080/01634372.2014.882466

Whittington, R., Hockenhull, J. C., McGuire, J., Leitner, M., Barr, W., Cherry, M. G., . . . Dickson, R. (2013). A systematic review of risk assessment strategies for populations at high risk of engaging in violent behaviour: Update 2002-8. *Health Technology Assessment, 17*(50), i-xiv, 1-128. doi:10.3310/hta17500

Wintrup, A. M., Coles, M., Webster, C. D., & Hart, S. D. (1994). *The predictive validity of the PCL-R in high-risk mentally disordered offenders*. Paper presented at the Canadian Psychological Association Annual Convention, Penticton, British Columbia.

Wintrup, A. M. (1996). *Assessing risk of violence in mentally disordered offenders with the HCR-20.* (Master of Arts), Simon Fraser University, Burnaby, British Columbia.

Vito, G. F., & Maahs, J. R. (2017). *Criminology: Theory, research, and policy* (4th ed.). Burlington, MA: Jones & Bartlett Learning.

Witt, K., Hawton, K., & Fazel, S. (2014). The relationship between suicide and violence in schizophrenia: Analysis of the Clinical Antipsychotic Trials of Intervention Effectiveness (CATIE) dataset. *Schizophrenia Research, 154*(1-3), 61-67. doi:10.1016/j.schres.2014.02.001

Witt, K., van Dorn, R., & Fazel, S. (2013). Risk factors for violence in psychosis: Systematic review and meta-regression analysis of 110 studies. *PloS One, 8*(2), e55942. doi:10.1371/journal.pone.0055942

Wittchen, H. U., & Jacobi, F. (2005). Size and burden of mental disorders in Europe--a critical review and appraisal of 27 studies. *European Neuropsychopharmacology, 15*(4), 357-376. doi:10.1016/j.euroneuro.2005.04.012

World Health Organization. (2002). *World report on violence and health*. Geneva, Switzerland: World Health Organization.

World Health Organization. (2017). The top 10 causes of death. Retrieved from http://www.who.int/mediacentre/factsheets/fs310/en/

World Health Organization. (2014). *Global status report on violence prevention 2014*. Geneva, Switerland: World Health Organization.

Yang, Y., & Raine, A. (2009). Prefrontal structural and functional brain imaging findings in antisocial, violent, and psychopathic individuals: A meta-analysis. *Psychiatry Research, 174*(2), 81-88. doi:10.1016/j.pscychresns.2009.03.012

Young, S., Moss, D., Sedgwick, O., Fridman, M., & Hodgkins, P. (2015). A meta-analysis of the prevalence of attention deficit hyperactivity disorder in incarcerated populations. *Psychological Medicine, 45*(2), 247-258. doi:10.1017/S0033291714000762

Yu, R., Geddes, J. R., & Fazel, S. (2012). Personality disorders, violence, and antisocial behavior: A systematic review and meta-regression analysis. *Journal of Personality Disorder, 26*(5), 775-792. doi:10.1521/pedi.2012.26.5.775

Zillman, D. (1988). Cognition-excitation interdependences in aggressive behavior. *Aggressive Behavior, 14*(1), 51-64. doi:10.1002/1098-2337(1988)14:1<51::AID-AB2480140107>3.0.CO;2-C